A STUDY ON POST MYOCARDIAL INFARCTION VENTRICULAR SEPTAL RUPTURE

Dissertation submitted in partial fulfilment
of the requirements for the degree of

M.Ch (CARDIO VASCULAR AND THORACIC SURGERY)
BRANCH - I

THE TAMILNADU DR. M.G.R. MEDICAL UNIVERSITY
CHENNAI

AUGUST 2007

CERTIFICATE

*This is to certify that, this dissertation titled "**POST MYOCARDIAL INFARCTION VENTRICULAR SEPTAL RUPTURE**" submitted by* **Dr. KULASEKARAN. M**, *appearing for M.Ch (Cardio Vascular and Thoracic Surgery) degree examination in August 2007 is a bonafide record of work done by him under my guidance and supervision.*

Porf. K.HARSHA VARDHAN, **M.S., M.Ch.,**
Professor and Head,
Department of Cardio Thoracic Surgery,
Madras Medical College,
Chennai - 600 003.

DEAN,
Madras Medical College,
Chennai - 600 003.

ACKNOWLEDGEMENT

I gratefully acknowledge and sincerely thank ***The Dean***, Madras Medical College, Chennai, for granting me permission to utilize the facilities of the institution for my study.

It is a proud privilege to express my heartfelt thanks and indebtedness to my enthusiastic and inspiring head of department ***Prof. K. Harshavardhan, M.S., M.Ch.***, for his Mastery guidance with supreme knowledge in conducting this study.

I express my sincere gratitude to ***Dr. A. Sugumar M.S., M.Ch.,*** who operated most of my study cases and taught me the art of Ventricular Septal Rupture Repair.

I am thankful to all my teachers ***Prof. L. Venkatachalapathy, Prof. T.S. Manoharan, Prof. S. Viswakumar, Prof. S. Vijayan, Dr. V.S. Manoharan, Dr. K. Rajavenkatesh, Dr. K. Sundaram, Dr. R.K. Sasankh, Dr. M. Nagarajan, Dr. A. Varadarajulu, Dr. J. Sivakumar,*** who guided me, taught me encouraged me and helped me during my study period in this institution.

I also would like to remember my fellow Post Graduates, Perfutionists and Staff Nurses for being a source of sustained strength.

I am extremely grateful to all our patients who had immense faith in us.

CONTENTS

SL.NO	TOPIC	PAGE NO
1.	Introduction	1
2.	Aims and Objectives	3
3.	Materials & Methods	4
4.	Review of Literature	5
5.	Observation & Results	44
6.	Conclusion	55
7.	Bibliography	
8.	Master Chart	

INTRODUCTION

Post infarction ventricular septal defect is a perforation of the muscular ventricular septum occurring in an area of acutely infarcted myocardium. Rupture of the ventricular septum following myocardial infarction is a relatively infrequent condition, which results in variable amounts of left-to-right shunting at the ventricular level and causes heart failure. The clinical presentation ranges from an asymptomatic murmur to cardiogenic shock. The first step in the evolution of surgical techniques to repair an acute Post infarction ventricular septal rupture involved differentiating the surgical treatment of these acquired lesions from the surgical approaches used to repair congenital ventricular septal defects, which are, for the most part, not applicable. Next, understanding the significance of differing anatomic locations of Post infarction ventricular septal defects led to innovations in terms of the location of the cardiotomy and the type of repair necessary to achieve a successful result in any given patient. Then, the gradual appreciation of different clinical courses pursued by patients after Post infarction ventricular septal rupture, both in terms of location of the defect and the degree of right ventricular functional impairment, led to an increased urgency

relative to the timing of surgical repair. More recently, improved results have been reported using the technique of endocardial patching with infarct exclusion, which may signify progress in the evolution of the surgical management of Post infarction ventricular septal defects. The incorporation of specific anatomic concepts of surgical repair and a better understanding of the physiologic basis ol the disease has led to an integrated approach to the patient that has improved salvage of patients suffering this catastrophic complication of acute myocardial infarction.

AIMS & OBJECTIVES

- To study incidence & demographics of post Myocardial Infarction ventricular Septal Rupture.
- To Study patterns of Coronary Artery Disease in post Myocardial Infarction ventricular Septal Rupture.
- To Analyse morphology of post Myocardial Infarction ventricular Septal Rupture
- To discuss current surgical principles of Ventricular Septal Rupture closure
- To Analyse factors influencing outcome after surgical treatment.

MATERIAL & METHODS

Between June 2002 to April 2007, 4247 patients with Acute Myocardial Infarction were treated at Coronary Care Unit at Government General Hospital, Chennai. 33 patients developed Post Myocardial Infarction ventricular Septal Rupture, 19 patients died during initial medical stabilization, 14 patients who survived and underwent surgical treatment were taken up for analytical study.

Incidence, Demographics, Risk factors, Clinical Presentation, Angiographic patterns, Ventricular Septal Defect Morphology, Surgical Techniques, and Out come were analysed.

LITERATURE REVIEW

HISTORY

In 1845 Latharn[2] described a post infarction ventricular septal rupture at autopsy, but it was not until 1923 that [3]Brunn first made the diagnosis antemortem.Sager[4] in 1934 added the 18th case to the world literature and established specific clinical criteria for diagnosis, stressing the association of post infarction septal rupture with coronary artery disease.

The treatment of this entity was medical and strictly palliative until 1956, when [5]Cooley et al performed the first surgical repair in a patient 9 weeks after the diagnosis of septal rupture. These first patients who underwent repairs in the early 1960s usually presented with congestive heart failure, having survived for more than a month after acute septal perforation[6,7]. The success of operation in these patients and the precipitous, acute course of other patients with this complication[8] gave rise to the belief that operative repair should be limited to patients surviving for 1 month or longer[6,9]. This, purportedly, allowed for scarring at the edges of the defect, which was thought to be crucial to the secure and long-lasting closure of the septal rupture[10,11]. In the late 1960s, more rapid recognition of

septal rupture following infarction led to the recommendation that operation be attempted earlier in patients who were hemodynamically deteriorating[1,3,12]. The use of improved prosthetic materials accompanied the successful surgical repair of defects from I to II days old, as reported by Allen[12] and Woodwark in 1966, Heimbecker[13] et al in 1968, and Iben[14] et al in 1969. Notable among these was a superb early study by Heimbecker et al of infarctectomy[13,15,16] and its clinical application to patients with post infarction ventricular septal defects. The surgical management of these patients was further refined by the inclusion of infarctectormy and aneurysmectomy[17,18] and the development of techniques to repair perforations in different areas of the septum[19,20,].

Over the last 15 years, it has become increasingly clear that in the majority of cases post infarction ventricular septal rupture constitutes a surgical emergency. More recently, improved surgical techniques, newer prosthetic materials, enhanced myocardial protection, and improved peri-operative mechanical and pharmacologic support have led to more favorable results in the surgical management of patients with post infarction septal rupture[21,22].

INCIDENCE & DEMOGRAPHICS

Post infarction ventricular septal defects complicate approximately 1% to 2% of cases of acute myocardial infarctions[23,24] and account for about 5% of early deaths after myocardial infarction. The average rime from infarction to rupture has been reported to be between 2 and 4 days, but it may be as short as a few hours or as long as 2 weeks[24-27]. These observations correlate well with the pathological findings, which demonstrate that necrotic tissue is most abundant and in growth of blood vessels and connective tissue is only beginning 4 to 21 days following a myocardial infarction [28,29]. Post infarction ventricular septal defects occur in men more often than women (3:2), but more women experience rupture than what would be expected from the incidence of coronary artery disease in women. The age of patients with this complication ranges from 44 to 81 years, with a mean of 62.5 years. However, there is some evidence that the average of post infarction age is increasing. The vast majority of patients who experience ventricular septal rupture do so after their initial infarction [30,31]. The overall incidence of post infarction ventricular septal rupture may have decreased slightly during the past decade as a result of aggressive pharmacologic treatment of ischemia and thrombolytic and interventional therapy in patients with evolving myocardial infarction as well as the prompt control of hypertension in these patients.

PATHOGENESIS

The infarct associated with septal rupture is transmural and generally quite extensive, involving, on average, 26% of the left ventricular wall in hearts with septal rupture, compared with only 15% in other acute infarctions[24]. In an autopsy study, [37]Cummings et al found that in patients with acute anterior or inferior infarctions, the amount of right ventricular infarction was much greater in the hearts with septal ruptures as compared to those without septal defects. Likewise, hearts with posterior septal rupture had more extensive left ventricular necrosis than did hearts with inferior infarctions and no septal defects.

Why certain hearts rupture and others do not is unclear at present. Slippage of myocytes during infarct expansion[38] may allow blood to dissect through the necrotic myocardium and enter either the right ventricle or pericardial space[19]. Hyaline degeneration of cardiomyocytes with subsequent fragmentation and enzymatic digestion may allow fissures to form, predisposing to rupture[40].

Post infarction ventricular septal defects are most commonly located in the anteroapical septum as the result of a full-thickness anterior infarction (in approximately 60% of cases). These anterior

septal ruptures are caused by anteroseptal myocardial infarction following occlusion of the left anterior descending artery. In about 20% to 40% of patients, the rupture occurs in the posterior septum following an inferoseptal infarction, which is usually due to occlusion of a dominant right coronary artery or, less frequently, a dominant circumflex artery[46]. Thus, ventricular septal perforations occur most frequently in 65-year-old men with single-vessel coronary disease and poor collateral flow who present 2 to 4 days following their first anterior myocardial infarction.

There are two types of rupture: simple, consisting of a direct through-and-through defect usually located anteriorly; and complex, consisting of a serpiginous dissection tract remote from the primary septal defect which is usually located inferiorly[41]. Multiple defects, which may develop within several days of each other, occur in 5% to 11% of cases and are probably due to infarct extension. Since a successful surgical outcome is related to adequacy of closure of septal defects, multiple defects must be sought preoperatively if possible, and certainly at the time of operative repair.

Of the small number of patients who survive the early period of ventricular septal rupture, 35% to 68% go on to develop ventricular

aneurysms[25, 34] through the process of ventricular remodeling[42]. This compares with a 12% incidence aneurysm formation in patients suffering an in infarction but no septal rupture[43], and probably relates to the size and transmural nature of the infarction associated with septal rupture. Post infarction septal rupture, especially in the posterior septum, may be accompanied by mitral valve regurgitation due to papillary muscle infarction or dysfunction. In approximately one third of cases of septal rupture, there is a degree of mitral insufficiency, usually functional in nature, secondary to left ventricular dysfunction with mitral annular dilation, which usually resolves with repair of the defect[33].

PATHOPHYSIOLOGY

The most important determinant of early outcome following post infarction ventricular septal rupture is the development of heart failure (left, right, or both). The associated cardiogenic shock leads to end-organ malperfusion, which may be irreversible. The degree to which heart failure develops depends on the size of the ventricular infarction and the magnitude of the left-to-right shunt. Left ventricular dysfunction due to extensive necrosis of the left ventricle is the primary determinant of heart failure and cardiogenic shock in patients with anterior septal rupture, while right ventricular

dysfunction secondary to extensive infarction of the right ventricle is the principal determinant of heart failure and cardiogenic shock in patients with posterior septal rupture. However, the development of congestive heart failure and cardiogenic shock in a patient with post infarction ventricular septal defects is not explained solely by the degree of damage sustained by the ventricle[46].

The magnitude of the left-to-right shunt is the other key variable in the development of hemodynamic compromise. With the opening of a ventricular septal defect, the heart is challenged by an increase in pulmonary blood flow, and a decrease in systemic blood flow as a portion of each stroke volume is diverted to the pulmonary circuit. As a consequence of the sudden increase in hemodynamic load imposed upon a heart already compromised by acute infarction, and possibly by a ventricular aneurysm, mitral valve dysfunction, or a combination of these problems, a severe low cardiac output state results. The normally compliant right ventricle is especially susceptible to failure in this circumstance[47,48]. Patients with posterior ventricular septal rupture and right ventricular dysfunction may display shunt reversal during diastole because the end-diastolic pressure in the right ventricle can be higher than in the left[49].

Ultimately, persistence of a low cardiac output state results in peripheral organ failure.

DIAGNOSIS

The typical presentation of a ventricular septal rupture is that of a patient who has suffered an acute myocardial infarction and who, after convalescing for a few days, develops a new systolic murmur, recurrent chest pain, and an abrupt deterioration in hemodynamics. The development of a loud systolic murmur, usually within the first week following an acute myocardial infarction, is the most consistent physical finding of post infarction ventricular septal rupture (present in over 90% of cases). The murmur is usually harsh, pan- systolic, and best heard at the left lower sternal border. The murmur is often associated with a palpable thrill. Depending on the location of the septal defect, the murmur may radiate to the left axilla, thereby mimicking mitral regurgitation[26]. Up to half of these patients experience post infarction chest pain in association with the appearance of the murmur.

Coincident with the onset of the murmur, there is usually an abrupt decline in the patient clinical course, with the onset of congestive failure and often cardiogenic shock. The findings of

cardiac failure that occur acutely in these patients are primarily the result of right-sided heart failure, with pulmonary edema being less prominent than that occurring in patients with acute mitral regurgitation due to ruptured papillary muscle[50].

The electrocardiographic findings in patients with acute septal rupture relate to the changes associated with antecedent anterior, inferior, posterior, or septal infarction. The localization of infarction by ECG correlates highly with the location of the associated sepal perforation. Daggett et al reviewed 55 patients with post infarction septal rupture, the location of the defect corresponded to the territory of transmural infarction as determined by ECG in all but three patients. Up to one third of patients develop some degree of atrioventricular conduction block (usually transient) that may precede rupture[51], but there is no pathognomonic prognostic indicator of impending perforation. The chest radiograph usually shows increased pulmonary vascularity consistent with pulmonary venous hypertension.

Advances in transthoracic and transesophageal cchocardiogra hy, especially color flow Doppler mapping, have revolutionized the diagnosis of both the presence and site of septal rupture[58-61].

Echocardiography can detect the defect, localize its site and size, determine right and left ventricular function, assess pulmonary artery and right ventricular pressures, and exclude coexisting mitral regurgitation or free wall rupture. Smyllie et al[60] reported a 100% specificity and 100% sensitivity when color Flow Doppler mapping was used to differentiate ventricular septal rupture from acute severe mitral regurgitation following acute myocardial infarction. It also correctly demonstrated the site of septal rupture in 41 of 42 patients. Widespread use of this technology has, for the most part, replaced thermodilution catheter insertion, which in outlying hospitals, where patients are often seen first, may be time consuming and difficult to accomplish. Indeed, the trend toward early surgical referral and prompt operative repair is at least partially explained by the more widespread use of color Doppler echocardiography for diagnosis in peripheral centers.

Angiographic evaluation of patients with post infarction ventricular rupture indicates that septal rupture is usually associated with complete occlusion rather than severe stenosis of a coronary artery[32]. On average, these patients have slightly less extensive coronary artery disease, as well as less developed septal collaterals than do other patients with coronary artery disease[33]. The lack of

collateral flow noted acutely may be secondary to anatomic configuration, edema, or associated arterial disease. Hill et al[34], in reviewing 19 cases of post infarction ventricular septal rupture, found single-vessel disease in 64%, double-vessel disease in 7%, and triple-vessel disease in 29%. However, the frequency of single-, double-, and triple-vessel coronary artery disease is more evenly distributed in other series[35].

NATURAL HISTORY

Reviews by Oyamada and Queen[65], Sanders et al[8], and Kirklin et al[66] reveal that nearly 25% of patients with post infarction septal rupture and no surgical intervention died within the first 24 hours, 50% died within 1 week, 65% within 2 weeks, 80% within 4 weeks; only 7% lived longer than one year. Lemery [67] et al reported that of 25 patients with post infarction ventricular septal defects treated medically, 19 died within one month. Thus, the risk of death following post infarction ventricular septal defect (VSD) is highest immediately after infarction and septal rupture, and then gradually declines, Interestingly, there are reports of spontaneous closure of small defects, though this is so rare that it would be unreasonable to manage a patient with the expectation of closure,

Recently, the SHOCK Trial (Should We Emergently Revascularize Occluded Coronaries in Cardiogenic Shock) provided intriguing data on the outcome of medically managed patients with shock and post infarction VSD[68], The multi-institutional study tracked 55 patients in cardiogenic shock from post infarction VSD. Rupture occurred a median of 16 hours after infarction, and the median time to the onset of shock was 7.3 hours. Twenty-four patients were managed medically; the remaining 31 patients comprised a high risk surgical group. There were only 7 survivors, of whom 6 had surgery to the repair the defect.

Despite the many advances in the nonoperative treatment of congestive heart failure and cardiogenic shock, including the intra-aortic balloon pump and a multitude of new inotropic agents and vasodilators, these do not supplant the need for operative intervention in these critically ill patients.

TIMING OF SURGERY

It has become clear that the early practice of waiting for several weeks after ventricular septal rupture before proceeding with surgery only selects out the small minority of patients in whom the hemodynamic insult is less severe and is better tolerated[19,35,69]. Likewise, it has also become clear that to manage most patients supportively, in hopes of deferring operation, is to deprive the great majority of those with post infarction ventricular septal rupture of the benefits of definitive surgery before irreversible damage due to peripheral organ ischemias occurred [62,70].

While Dagett[21] and others[69] have advocated early surgery since the middle of the 1970s, some continue to prefer to defer operation in patients who are easily supported and exhibit no further hemodynamic deterioration[71,72]. Persistence of congestive heart failure or marginal stabilization with rising blood urea nitrogen (BUN) and borderline urine output necessitate aggressive therapy and prompt operation. The routine use of the intra-aortic balloon pump, whenever technically feasible, frequently results in transient reversal of the hemodynamic deterioration. This period of stability often makes it possible to complete left heart catheterization before

proceeding to operation but should not significantly delay definitive surgical treatment. Patients with septal rupture rarely die of cardiac failure per se, but rather of end-organ failure as a consequence of shock. Shortening the duration of shock by operating early is the only therapeutic solution for this group of patients and can yield dramatic results[73].

Patients in cardiogenic shock represent a true surgical emergency requiring immediate operative repair. Because deaths in these patients result from multisystem failure secondary to organ hypoperfusion, delay in operative repair for patients in cardiogenic shock represents a “failed therapeutic strategy.” Those few patients who are completely stable, with no clinical deterioration, and who require no hemodynamic support, can undergo operative repair when convenient during that hospitalization. The large group of patients who are in an intermediate position between those with shock and those in stable condition should be operated on early (usually within 12 to 24 hours) after appropriate preoperative evaluation. Since the group of patients in stable condition constitutes 5% or less of the total population of patients with post infarction ventricular septal rupture, the overwhelming majority of patients require prompt surgical treatment.

Rarely, because of a delayed referral, a patient will be seen for surgical therapy who is already in a state of multisystem failure or has developed septic complications. Such a patient is unlikely to survive an emergency operation and thus may benefit from prolonged support with an intra-aortic balloon pump before an attempted operative repair. Baillot[72] et al have reported individual successes with such an approach.

PREOPERATIVE MANAGEMENT

Because the natural course of the disease in unoperated patients is so dismal, the diagnosis of post infarction ventricular septal rupture can be regarded as its own indication for operation. Preoperative management is directed towards stabilization of the hemodynamic condition so that peripheral organ perfusion can be best maintained while any further diagnostic studies are obtained and while deciding on the optimal time for surgical intervention. Although the early clinical course of patients with post infarction ventricular septal rupture can be quite variable, 50% to 60% present with severe congestive heart failure and a low cardiac output state requiring intensive therapy[74].

The goals of preoperative management are to: (1) reduce the systemic vascular resistance, and thus the left-to-right shunt; (2) maintain cardiac output and arterial pressure to ensure peripheral organ perfusion; and (3) maintain or improve coronary artery blood flow This is best accomplished by the intra-aortic balloon pump (IABP). Counterpulsation reduces left ventricular afterload, thereby increasing cardiac output arid decreasing the left-to-right shunt, as reported by Gold et al[75] in 1973. In addition, IABP support is associated with decreased myocardial oxygen consumption, as well as improved myocardial and peripheral organ perfusion. Although counterpulsation produces an overall improvement in the patient's condition, a complete correction of the hemodynamic picture cannot be obtained[76]. Peak improvement occurs within 24 hours and no further benefit has been observed with prolonged balloon pumping[71]. Pharmacologic therapy with inotropic agents and diuretics should be instituted promptly. The addition of vasodilators (i.e., sodium nitroprusside or intravenous nitroglycerine) makes good theoretical sense, because it can decrease the left-to-right shunting associated with the mechanical defect, and thus increase cardiac output. However, these effects are often associated with a marked fall in mean arterial blood pressure and reduced coronary perfusion, both

poorly tolerated in these critically ill patients. It must he stressed that pharmacologic therapy is intended primarily to support the patient in preparation for operation and should not in any way delay urgent operation in the critically ill patient. Patients with post infarction septal rupture are directly admitted to the surgical intensive care unit rather than to the coronary care or medical intensive care unit.

OPERATIVE TECHNIQUES

Approach:

The first repair by Cooley et a1[5] of an acquired ventricular septal defect was accomplished using an approach through the right ventricle with incision of the right ventricular outflow tract. This approach, which was adapted from surgical techniques for closure of congenital ventricular septal defects, proved to be disadvantageous for many reasons. Exposure of the defect was frequently less than optimal, particularly for defects located in the apical septum. It involved unnecessary injury to normal right ventricular muscle and interruption of collaterals from the right coronary artery. Finally, it failed to eliminate the paradoxical bulging segment of infarcted left ventricular wall. Subsequently, Heimbecker et al[13] introduced, and others adopted [16,25,81], a left-sided approach (left ventriculotomy) with incision through the area of infarction. Such an approach frequently incorporates infarctectomy and aneurysmectomy, together with repair of septal rupture.

Repair of Post infarction VSD can be accomplished through a right atrial approach. The potential advantage of this technique is that an incision in the LV myocardium is avoided. Experience with

this procedure is limited.[125,126] Massetti and colleagues have reported the largest series (12 patients). There were three early deaths (25%) and one late death. One patient required reoperation for a residual defect 3 months postoperatively, and one had a small residual left-to-right shunt. Of the eight surviving patients who were followed for a mean of 5 years, three were NYHA functional class I, four were class II, and one was class III.

PRINCIPLES OF REPAIR OF POST INFARCTION VENTRICULAR SEPTAL DEFECTS

1. Expeditious establishment of total cardiopulmonary bypass with moderate hypothermia and meticulous attention to myocardial protection.
2. Transinfarct approach to ventricular septal defect with the Site of ventriculotomy determined by the location of the transmural infarction.
3. Thorough trimming of the left ventricular margins of the infarct back to viable muscle to prevent delayed rupture of the closure.
4. Conservative trimming of the right ventricular muscle as required for complete visualization of the margins of the defect.
5. Inspection of the left ventricular papillary muscles and concomitant replacement of the mitral valve only if there is frank papillary muscular rupture.
6. Closure of the septal defect without tension, which in most instances will require the use of prosthetic material.
7. Closure of the infarctectomy without tension with generous use of prosthetic material as indicated, and epicardial placement of the patch to the free wall to avoid strain on the friable endocardial tissue.
8. Buttressing of the suture lines with pledgets or strips of Teflon felt or similar material to prevent sutures from cutting through friable muscle.

GENERAL TECHNIQUES

Patients are anesthetized using a fentanyl-based regimen. Pancuronium is selected as the muscle relaxant so as to prevent bradycardia. Pulmonary bed vasodilators such as dobutamine are avoided to minimize the left-to-right shunt fraction. Preoperative broadspectrum antibiotics are given as prosthetic material may be left in the patient.

Cardiopulmonary bypass is accomplished, with bicaval venous drainage. Systemic cooling to 25°C is employed. Cardiac standstill is achieved with cold, oxygenated, dilute blood cardioplegia using antegrade induction followed. by retrograde perfusion via the coronary sinus. as total coronary occlusion is anticipated in most cases. A total of 1200 to 2000 ml of cardiqplegia solution is delivered depending on the size of the heart and the degree of hypertrophy. Patients with multivessel coronary disease and critical coronary stenoses are revascularized before opening the heart in order to optimize myocardial protection. Saphenous vein rather than the left internal mammary artery is utilised as graft.

APICAL SEPTAL RUPTURE

The technique of apical amputation was described by Daggett et al in 1970[16].

An incision is made through the infarcted apex of the left ventricle.

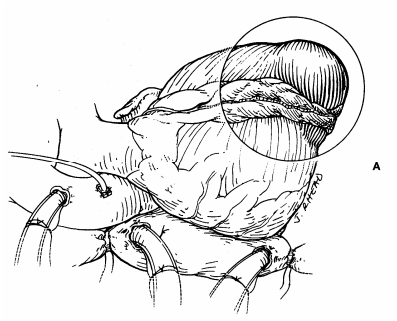
Excision of the necrotic myocardium back to healthy muscle results in amputation of the apical portion of the left ventricle, right ventricle, and septum. The remaining apical portions of the left and right ventricle free walls are then approximated to the apical septum.

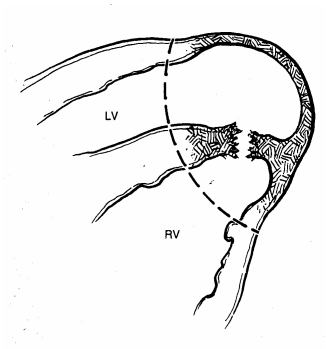
This is accomplished by means of a row of interrupted mattress sutures passed sequentially through a buttressing strip of Teflon felt, the left ventricular wall, a second strip of felt, the interventricular septum, a third strip of felt, the right ventricular wall, and a fourth strip of felt.

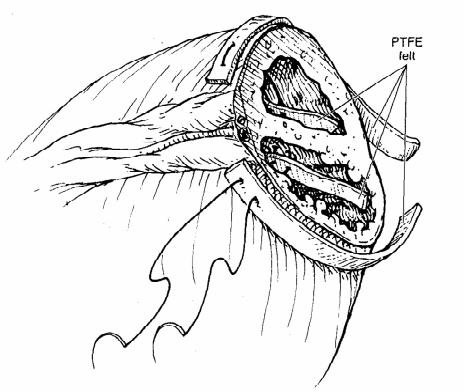
After all sutures have been tied, the closure is reinforced with an additional over-and-over suture, as in ventricular aneurysm repair, to insure hemostasis of the ventriculotomy closure.

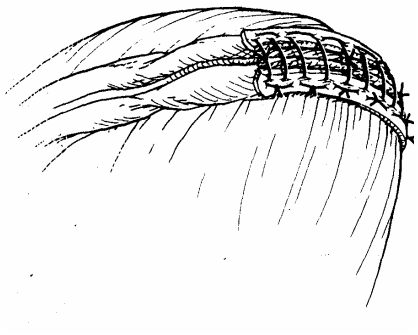
APICAL SEPTAL RUPTURE:

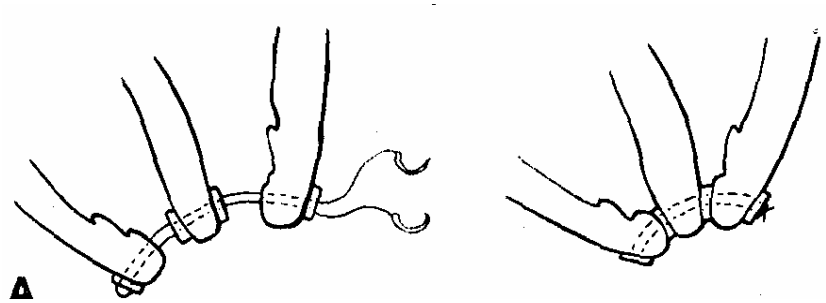
Daggetts Amputation Technique











ANTERIOR SEPTAL RUPTURE

SHUMAKER PLICATION TECHNIQUE:

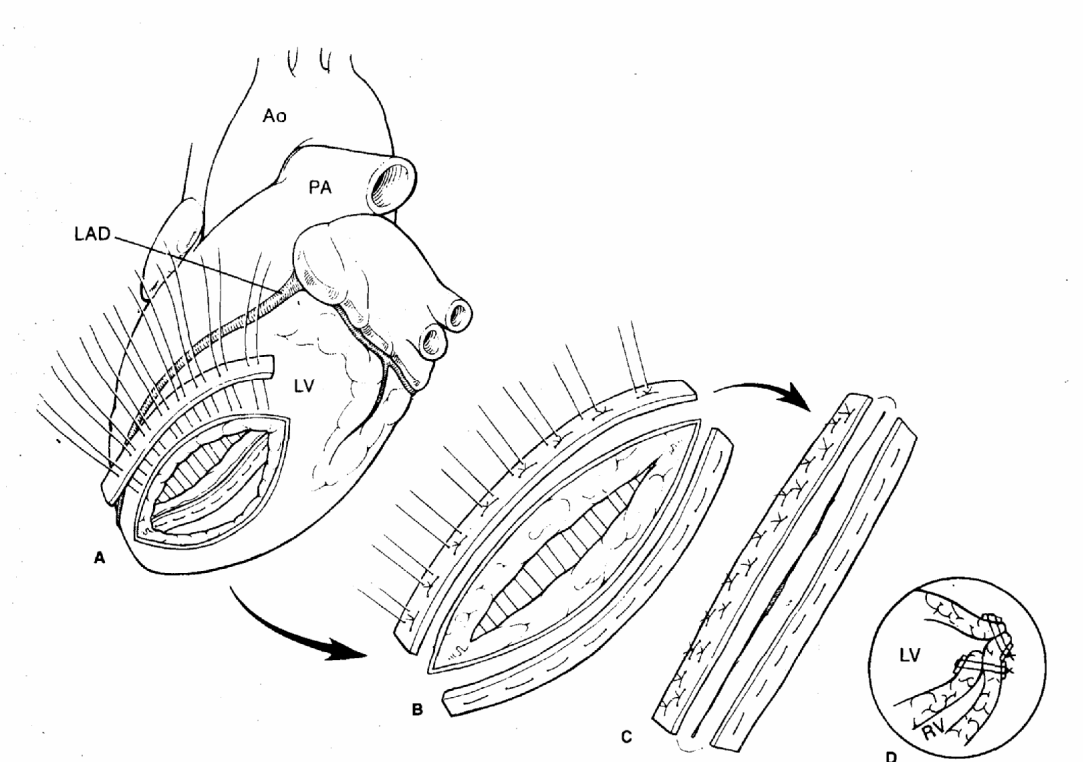
Small defects beneath anterior infarcts can be closed by the technique of plication as suggested by Shumaker.

This involves approximation of the free anterior edge of the septum to the right ventricular free wall using mattress sutures over strips of felt. The transinfarct incision is then closed with a second row of mattress sutures buttressed with strips of felt. An over-and over running suture completes the ventriculotomy closure.

The disadvantage with this techniques in the tension at suture line in already friable myocardium suitable only for small chronic VSR located anteriorly

ANTERIOR SEPTAL RUPTURE

Shumaker Plication Technique:



ANTERIOR SEPTAL RUPTURE

PATCH CLOSURE:

Most anterior defects require closure with a prosthetic patch in order to avoid tension that could lead to disruption of the repair.

After debridement of necrotic septum and left ventricular muscle, a series of pledgeted interrupted mattress sutures are placed around the perimeter of the defect.

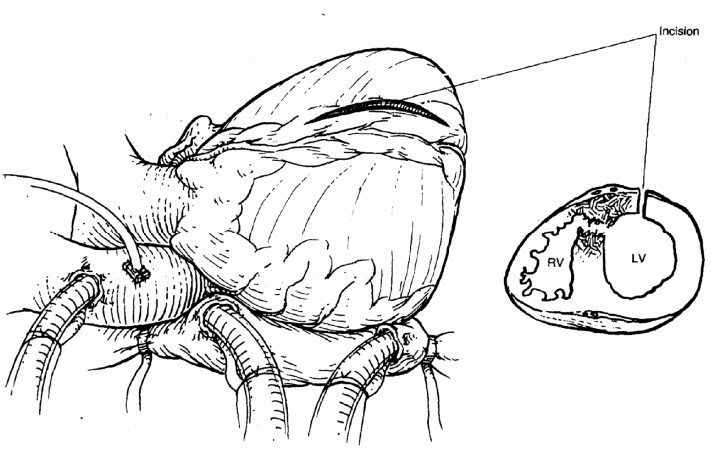
Along the posterior aspect of the defect, sutures are passed through the sepum from right side to left. Along the anterior edge of the defect, sutures are passed from the epicardial surface of the right ventricle to the endocardial surface.

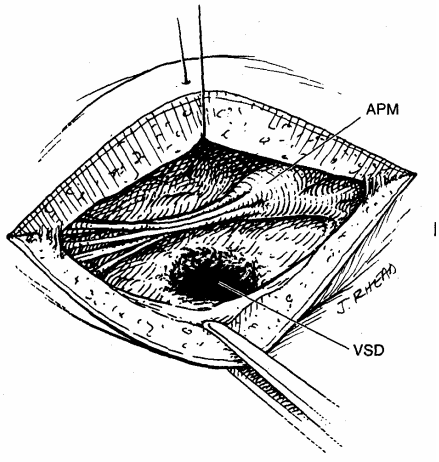
All sutures are placed before the patch is inserted, and then passed through the edge of a synthetic patch, which is seated on the left side of the septum.

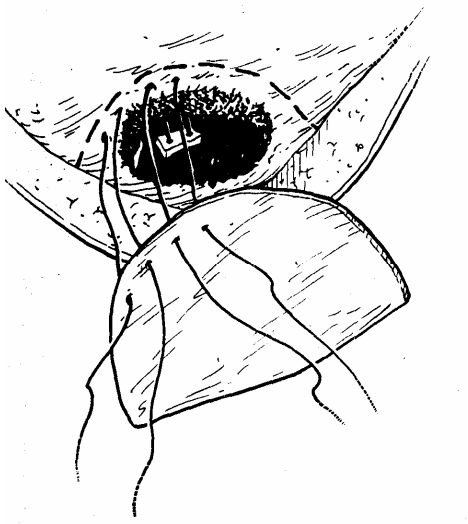
Each suture is then passed through an additional pledget and all are tied. Additional pledgets on the left ventricular side overlying the patch to cushion each suture as it is tied down to prevent cutting through the friable muscle. The edges of the ventriculotomy are then approximated by a two-layer closure consisting of interrupted mattress sutures passed through buttressing strips of Teflon felt (or glutaraldehyde preserved bovine pericardium) and a final over-and-over running suture.

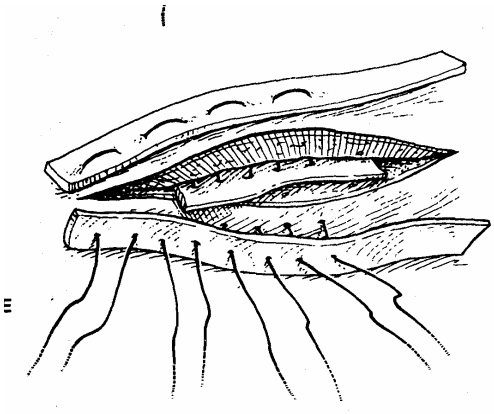
ANTERIOR SEPTAL RUPTURE

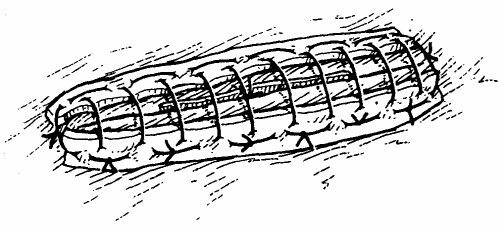
PATCH CLOSURE:









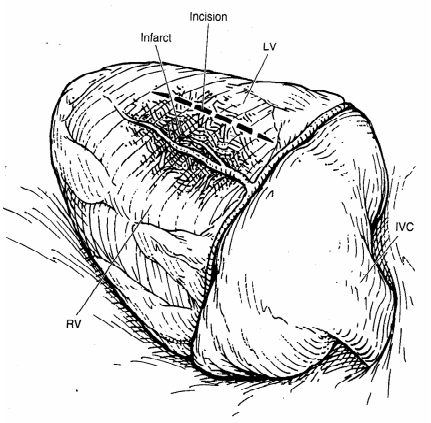


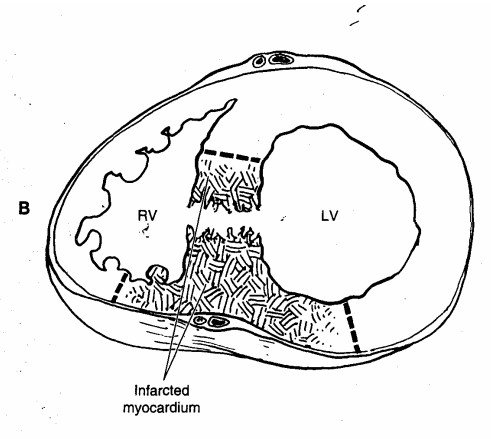
POSTERIOR / INFERIOR SEPTAL RUPTURE

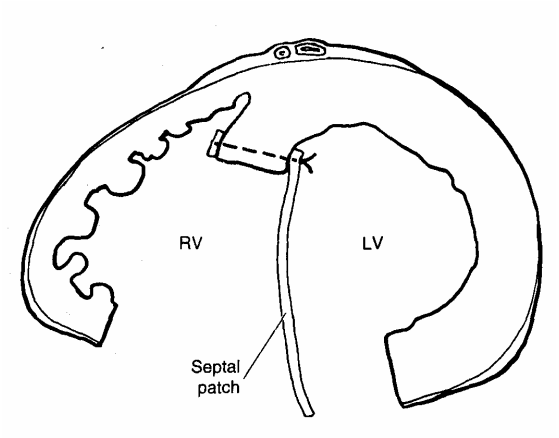
Closure of inferopostcrior septal defects, which result from transmural infarction in the distribution of the posterior descending artery, has posed the greatest technical challenge. Early attempts at primary closure of these defects by simple plication techniques similar to those used in the repair of anterior defects were frequently unsuccessful because of the sutures tearing out of soft, friable myocardium that had been closed under tension. This resulted in either reopening of the defect or catastrophic disruption of the infarctectomy closure.

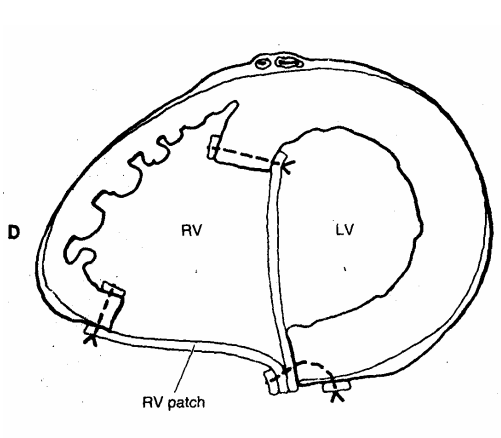
Use of the following techniques has been associated with an improved operative survival. After the establishment of bypass with bicaval cannulation, the left side of the heart is vented via the right superior pulmonary vein. The heart is retracted out of the pericardial well as for bypass to the posterior descending coronary artery. The margins of the defect may involve the inferior aspects of both ventricles, or of the left ventricle only. A transinfarct incision is made in the left ventricle, and the left ventricular portion of the infarct is excised, exposing the septal defect. The left ventricular papillary muscles are inspected. Only if there is frank papillary muscle rupture

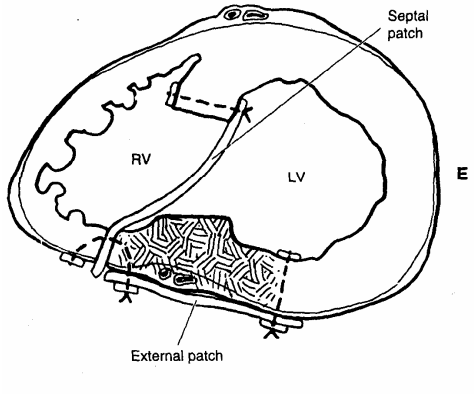
POSTERIOR SEPTAL RUPTURE – PATCH CLOSURE

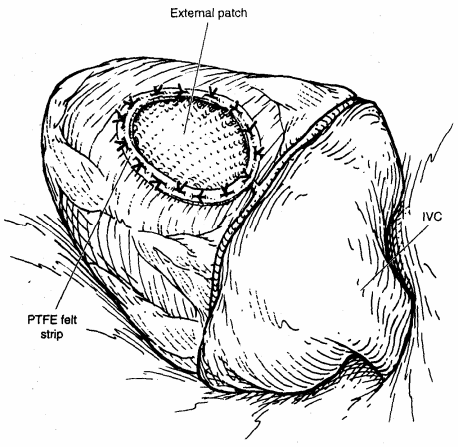












is mitral valve replacement performed. When it is indicated mitral valve replacement performed through a separate conventional left atrial incision, to avoid trauma to the friable ventricular muscle. After all infarcted left ventricular muscle has been excised, a less aggressive debridement of the right ventricle is accomplished, with the goal of resecting only as much muscle as is necessary to afford complete visualization of the defect(s). Using this technique delayed rupture of the right ventricle has not been a problem. If the posterior septum has cracked or split from the adjacent ventricular free wall without loss of a great deal of septal tissue, then the septal rim of the posterior defect may be approximated to the edge of the diaphragmatic right ventricular free wall using mattress sutures buttressed with strips of Teflon felt or bovine pericardium. Larger posterior defects require patch closure. Pledgeted mattress sutures are placed from the right side of the septum and from the epicardial side of the right ventricular free wall. All sutures are passed through the perimeter of the patch and then through additional pledgets, and are then tied. Thus, as in closure of large anterior defects, the patch is secured on the left ventricular side of the septum. Direct closure of the remaining infarctectomy is rarely possible because of tension

required to pull together the edges of the gaping defect. A prosthetic patch is generally required.

Pledgeted mattress sutures are passed out through the margin of the infarctectomy (endocardium to epicardium) and then through the patch, which is seated on the epicardial surface of the heart. After each suture is passed through an additional pledget, all sutures are tied. The cross-sectional view of the completed repair illustrates the restoration of relatively normal ventricular geometry, which is accomplished by the use of appropriately sized prosthetic patches.

ENDOCARDIAL PATCH REPAIR WITH INFARCT EXCLUSION

DAVIDS TECHNIQUE

The concept that the preservation of left ventricular geometry plays a crucial role in the preservation of left ventricular function[90] has laid the groundwork for a recent evolution in the surgical approach to post infarction ventricular septal defects - the technique of endocardial patch repair of post infarction ventricular septal defects described - by David[81,84], Cooley [91,92], and then by Ross[93] in the early 1990s. This operative technique, which is an application to ventricular septal rupture repair of Dor's technique of ventricular endoaneurysmorrhaphy[90], involves intracavitary placement of an endocardial patch to exclude infarcted myocardium while maintaining ventricular geometry. Thus, instead of closing the septal defect, it is simply excluded from the high-pressure zone of the left ventricle.

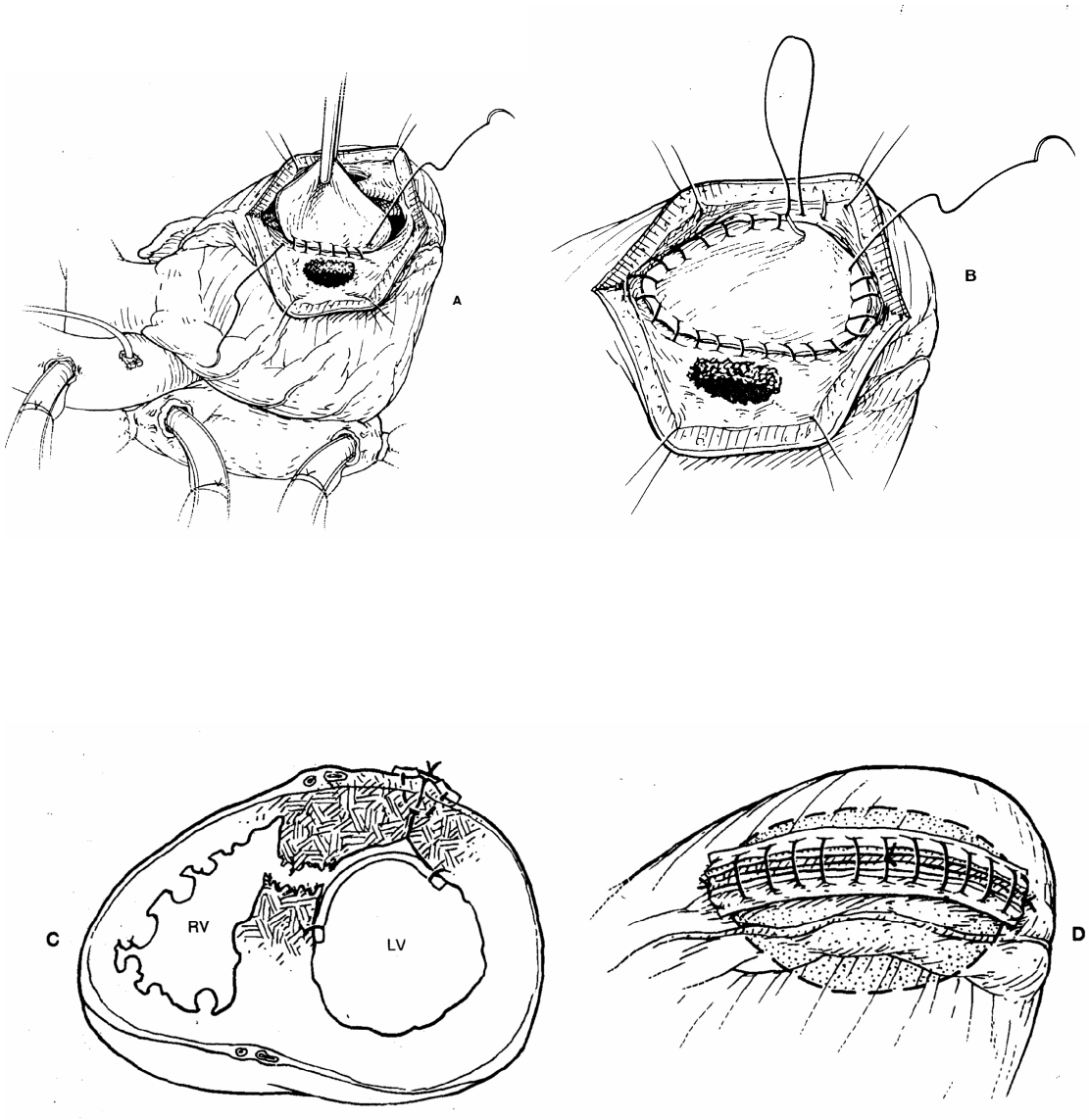
There are several theoretical advantages in the technique of infarct exclusion. (1) It does not require resection of myocardium; excessive resection results in depression of ventricular function and insufficient resection predisposes to recurrence of septal rupture. (2) It maintains ventricular geometry, which enhances ventricular function. (3) It avoids tension on friable muscle, which may diminish postoperative bleeding.

ANTERIOR SEPTAL RUPTURE INFARCT EXCLUSION TECHNIQUE

In patients with anterior septal rupture, the interventricular septum is exposed via a left ventriculotomy, which is made through the infarcted anterolateral wall starting at the apex and extending proximally parallel to but 1 to 2 cm away from the anterior descending artery. Stay sutures are passed through the margins of the ventriculotomy to aid in the exposure of the infarcted septum. The septal defect is located and the margins of the infarcted muscle identified. A glutaraldehyde-fixed bovine pericardial patch is tailored to the shape of the left ventricular infarction as seen from the endocardium but 1 to 2 cm larger. The patch is usually oval and measures approximately 4 x 6 cm in most patients. The pericardial patch is then sutured to healthy endocardium all around the infarct. Suturing begins in the lowest and most proximal part of the noninfarcted endocardium of the septum with a continuous 3-0 polypropylene suture. Interrupted mattress sutures with felt pledgets may be used to reinforce the repair. The patch is also sutured to the noninfarcted endocardium of the anterolateral ventricular wall. The stitches should be inserted 5 to 7 mm deep in the muscle and 4 to 5 mm apart. The stitches in the patch should be at least 5 to 7 mm

ANTERIOR SEPTAL RUPTURE

INFARCT EXCLUSION TECHNIQUE



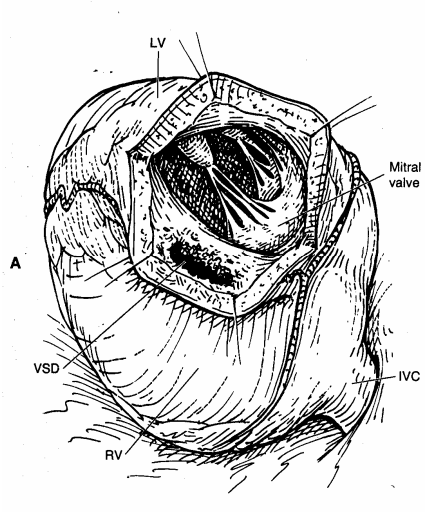
from its free margin so as to allow the patch to cover the area between the entrance and exit of the suture in the myocardium. This technique minimizes the risk of tearing muscle as the suture is pulled taut. If the infarct involves the base of the anterior papillary muscle, the suture is brought outside of the heart and buttressed on a strip of bovine pericardium or Teflon felt applied to the epicardial surface of the left ventricle. Once the patch is completely secured to the endocardium of the left ventricle, the left ventricular cavity becomes largely excluded from the infarcted myocardiurn. The ventriculotomy is closed in two layers over two strips of bovine pericardium or Teflon felt using 2.0 or 3-0 polypropylene sutures. No infarctectomy is performed unless the necrotic muscle along the ventriculotomy is sloughing at the time of its closure, and even then it is minimized, since infarcted muscle will not be exposed to left ventricular pressures when the heart begins to work. Alternatively, sutures can be passed through the ventricular free wall and through a tailored external patch of Teflon or pericardium.

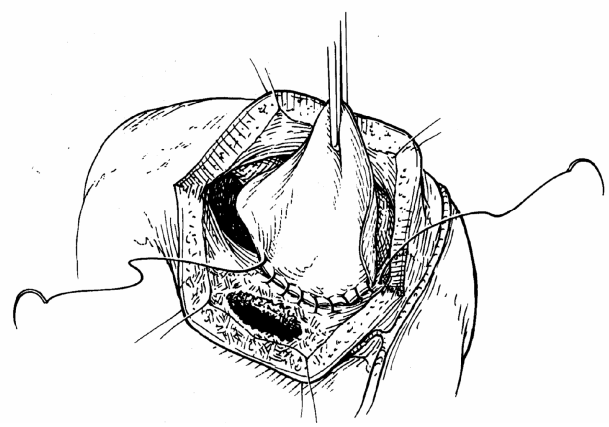
POSTERIOR SEPTAL RUPTURE

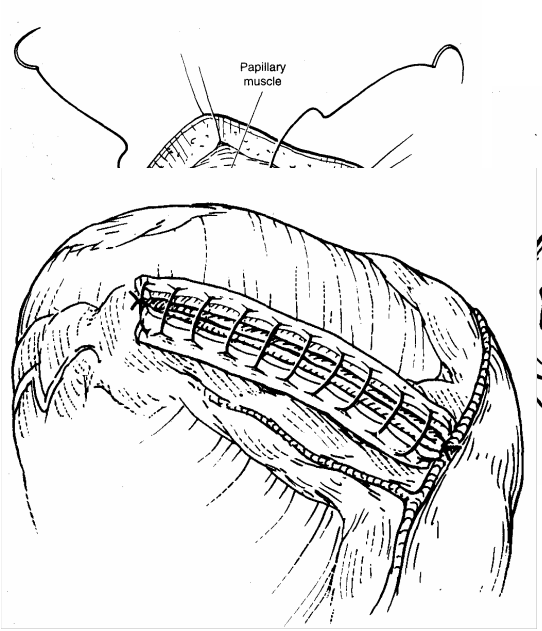
INFARCT EXCLUSION TECHNIQUE

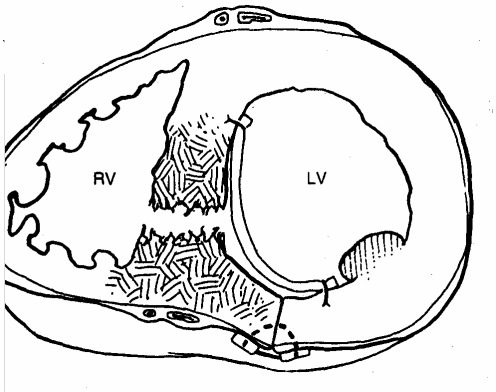
In patients with posterior septal defects, an incision is made in the inferior wall of the left ventricle 1 or 2 mm from the posterior descending artery. This incision is started at the midportion of the inferior wall and extended proximally toward the mitral annulus and distally toward the apex of the ventricle. Care is taken to avoid damage to the posterolateral papillary muscle. Stay sutures are passed through the fat pad of the apex of the ventricle and margins of the ventriculotomy to facilitate exposure of the ventricular cavity. In most cases, the rupture is found in the proximal half of the posterior septum and the posteromedial papillary muscle is involved by the infarction. A bovine pericardial patch is tailored in a triangular shape of approximately 4x7 cm in most patients. The base of the triangular-shaped patch is sutured to the fibrous annulus of the mitral valve with a continuous 3-0 polypropylene suture starting at a point corresponding to the level of the posteromedial papillary muscle and moving medially toward the septum until the noninfarcted endocardium is reached. At that level, the suture is interrupted and

POSTERIOR SEPTAL RUPTURE INFARCT EXCLUSION TECHNIQUE









any excess patch material trimmed. The medial margin of the triangular-shaped patch is sewn to healthy septal endocardium with a continuous 3-0 or 4-0 polypropylene suture taking bites the same size as described for anterior defects. In this area of the septum, reinforcing pledgeted sutures may be required. The lateral side of the patch is sutured to the posterior wall of the left ventricle along a line corresponding to the medial margin of the base of the posteromedial papillary muscle. Because the Posterior wall of the left ventricle is infarcted, it is usually necessary to use full-thickness bites and anchor the sutures on a strip of pericardium or TeflonfeIt applied on the epicardial surface of the posterior wall of the left ventricle right at the level of the posteromedial papillary muscle insertion. Once the patch is completely sutured to the mitral valve annulus, the endocardium of the interventricular septum, and the full thickness of the posterior wall, the ventriculotomy is closed in two layers of full thickness sutures buttressed on strips of pericardium or Teflon felt. The infarcted right ventricular wall is left undisturbed. If the posteromedial papillary muscle is ruptured, mitral valve replacement is necessary.

SIMULTANEOUS MYOCARDIAL REVASCULARIZATION

There has been controversy in the literature concerning the advantages and disadvantages of concurrent coronary artery grafting in patients undergoing emergent repair of post infarction ventricular septal rupture. Some have argued that revascularization provides no survival benefit and subjects patients to preoperative left heart catheterization, a time-consuming and potentially dangerous diagnostic procedure [63,64]. Loisance[105] et al base their policy of not revascularizing patients with post infarction septal ruptures on the fact that none of their 20 long-term survivors (5 of whom were bypassed) had incapacitating angina or recurrent myocardial infarction. Piwnica[106] et al reported a series of 28 survivors of early operative closure of Post infarction ventricular septal rupture, among whom only one had coronary artery grafting. Among the 24 patients for whom follow-up was complete, there were only 2 late deaths of cardiac origin. However, it is not clear from their report what the impact of associated coronary artery disease (revascularized or not) may have been on the course of the other 32 patients who did not survive operation.

Some groups use left heart catheterization and coronary bypassing selectively. Davies et al[30] found that of 60 long-term survivors (median 70 months; range 1 to 174 months), only five patients developed exertional angina during follow-up and none required revascularization. Their current policy is to avoid left heart catheterization on patients in whom an acquired septal defect is suspected to be a consequence of their first anterior infarction, provided that the patient has no history of angina or electrocardiographic evidence of previous infarction in another territory. This approach is also based on the findings that multivessel disease is much less prevalent in those with an apical septal rupture as a result of anterior infarction.

Degett and others[93,107] tend to employ coronary revascularization with increasing frequency. Their policy is to place aortocoronary grafts to principal epicardial coronary arteries that have severe proximal stenoses. In a more recent review, the effect of bypass was less dramatic, not achieving statistical significance. Nevertheless, there is no information that would suggest any negative impact of bypass grafting, and bypasses can be performed routinely when the clinical presentation permits catheterization.

WEANING FROM CARDIOPULMONARY BYPASS

The two most common problems encountered in separating from bypass following repair of a Post infarction ventricular septal defect are low cardiac output and bleeding.

In patient with low cardiac output, an IABP should be inserted before surgery. If not, one should be inserted in the operating room, especially if the low output state is secondary to left ventricular dysfunction. Also, IABP may benefit patients with right ventricular failure by improving right coronary artery blood flow due to diastolic augmentation. Intravenous milrinone, a phosphodiesterase inhibitor, is very effective in reversing low output slates secondary to left ventricular dysfunction. Milrinone possesses a balance of inotropic and vasodilatory properties that together produce an increase in cardiac output and reduction in right and left filling pressures and systemic vascular resistance. It is less arrhythmogenic than dobutamine, causes less hypotension than amrinone, and is not associated with throrombocytopenia.

Posterior defects are commonly associated with mitral regurgitation and right heart dysfunction secondary to extensive right ventricular infarction. Management of right heart failure is aimed at reducing right ventricular after load while maintaining systemic pressure. Initial steps to manage right ventricular dysfunction include volume loading, inotropc support, and correction of acidosis, hypoxemia, and hypercarbia.

To prevent postpump coagulopathy, antifibrinolytic therapy with either aprotinin or ε-aminocaproic acid (Amicar) is used before commencing cardiopulmonary bypass.

Half-dose aprotinin is administered by first giving an intravenous test dose of 10,000 KIU over 10 minutes (before administering blockers), and then loading patients with 1 million KIU over 20 minutes prior to bypass. Another 1 million KIU is given in the pump prime, and then 250,000 KIU/hr is administered for the duration of the surgery Heparm is managed in the usual fashion with activated clotting times (ACT), but kaolin, not Celite, is used as the ACT activator. Since controversy surrounds the issue of increased renal dysfunction and perioperative thrombotic events in patients receiving aprotinin, Amicar is used in patients who (1) require aortocoronary bypasses, (2) are diabetic, or (3) have known renal dysfunction.

Amicar is administered by loading patients with 10g prior to commencing bypass and then adding another 10g to the pump prime. During the procedure Amicar is continuously infused at 1g/h for the duration of surgery We avoid giving over 30g of Amicar.

Postpump suture line bleeding may be reduced by application of a fibrin sealant to the ventricular septum around the septal defect prior to formal repair. Biological glue may be effective in controlling bleeding suture lines following repair. As a last resort, Baldwin and Cooley have suggested insertion of a left ventricular assist device solely as an adjunct to the repair of friable or damaged myocardium to reduce left ventricular distension and thus control bleeding.

POSTOPERATIVE CARE

Early postoperative diuresis and positive end-expiratory pressure ventilation are used to decrease the arterial-alveolar gradient induced by the increased extravascular pulmonary water associated with cardiopulmonary bypass. Once the patient has warmed, intravenous infusion of Lasix combined with mannitol (1g of Lasix in 400cc of 20% mannitol is used at a rate of 1 to 20 cc per hour to keep the urine output greater than 100 cc per hour. If renal function has been compromised preoperatively, continuous venovenous hemofiltration (CVVH) is employed postoperatively.

Intractable postoperative venfticular arrhythmias secondary to reperfusion injury is controlled using intravenous amiodarone (10-20mg/kg over 24 hours).

OPERATIVE MORTALITY AND RISK FACTORS FOR DEATH

The most important predictor of operative mortality was preoperative hemodynaniic instability[119]. Patients in this group are usually in cardiogenic shock, are emergency cases, are on inotropic support, and usually have intra-aortic balloon pumps.

Additional risk factors for early and late death include the presence of left main coronary artery disease, previous myocardial infarction, renal dysfunction, and right heart failure. Other factors have been found to increase the risk of early death. Posterior location of the septal rupture has been associated with an increased operative mortality[47,49]. This has been attributed to a more technically difficult repair[120], to the increased risk of associated mitral regurgitation, and to associated right ventricular dysfunction that is an independent predictor of early mortality following posterior infarction. A short time interval between infarction and operation selects for sicker patients unable to be managed medically. Patient age has also been associated with an increased early mortality [122].

Proximal location of the septal defect (not posterior) was the main predictor for cardiogenic shock, which in turn was the strongest determinant of early mortality (34.3% vs. 16.7% for distal septal

defects)[119]. This relationship results from the fact that proximal septal defects are associated with larger infarctions.

Regardless of the technique, the most common cause of death following repair of acute Post infarction ventricular septal defect was low cardiac output syndrome (52%). Technical failures, most commonly recurrent or residual VSD but including bleeding, were the second most common (23%). Other causes of death include sepsis (17%), recurrent infarction (9%), cerebrovascular complications (4%), and intractable ventricular arrhythmias.

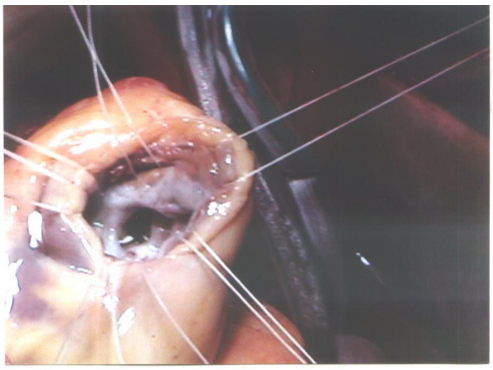
LONG - TERM RESULTS

Long-term results have been favorable as regards both mortality risk and functional rehabilitation. Actuarial survival at 5 years for most recent series generally ranges between 40% and 60%. Due to the overall high 1isk of the operation, it is rewarding to note that hospital survivors enjoy excellent longevity, with 1-, 5-, and 10-year survival of 91%, 70%, and 37%.

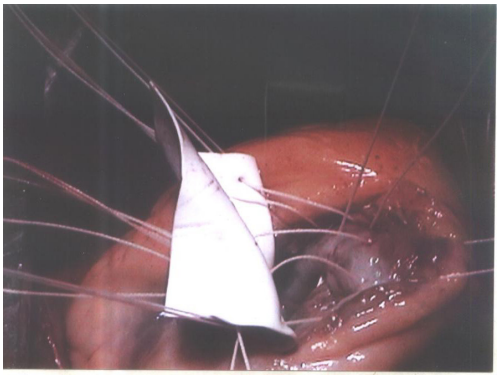
Gaudiani et al[74] reported long-term results using an early operative approach. In their series, 88% of hospital survivors were alive at 5 years, with 74% of survivors in NYHA functional class I and 21% of survivors in class II. In the series of patients reported by Piwnica et al[105], there were 20 long-term survivors, of whom 8 were in class I and 12 were in class II. David et a1[45] have reported a 66%, 6-year survival rate in patients operated on since 1980. Finally, Davies et a1 [30] reported 5-, 10-, and 14-year survivals of 69%, 50%, and 37%. Eighty-two percent of patients were in NYHA functional Class I or II.

ANTERIOR SEPTAL RUPTURE

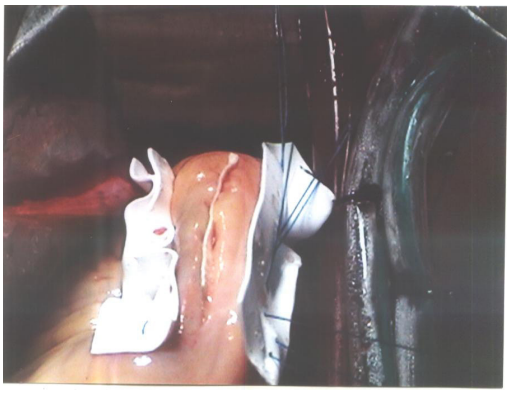
a.



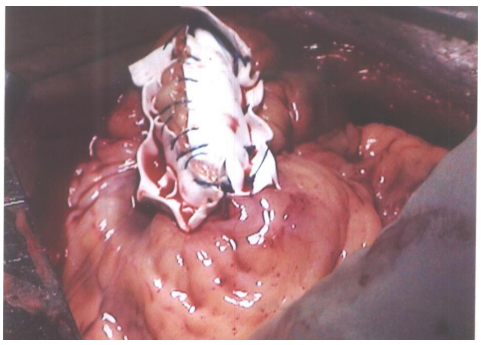
b.



c.

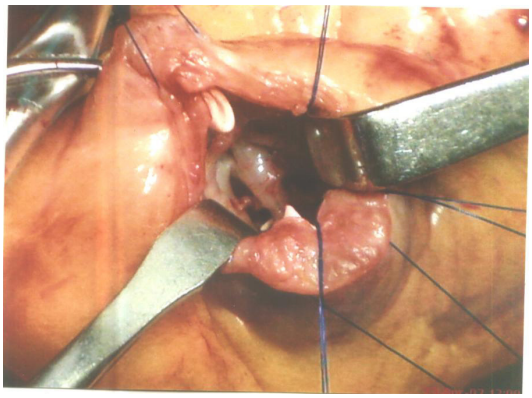


d.

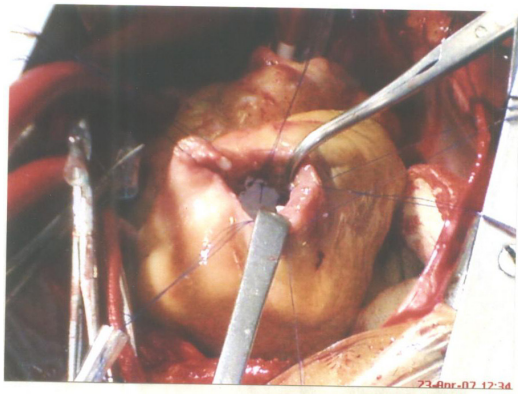


MULTIPLE VSR

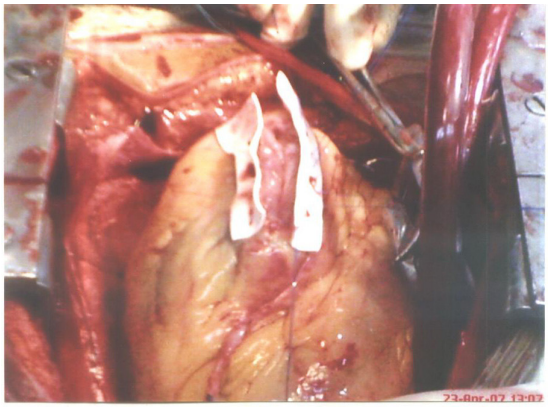
a.



b.

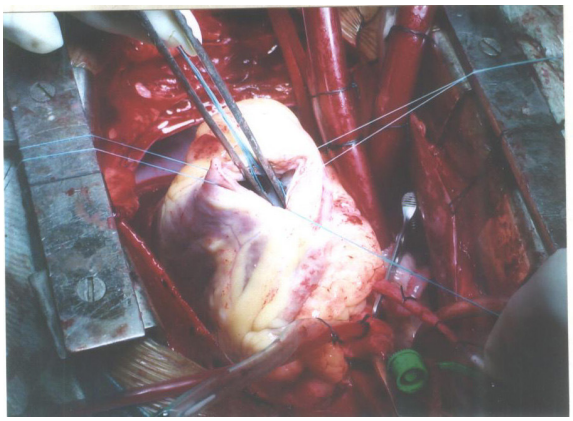


c.

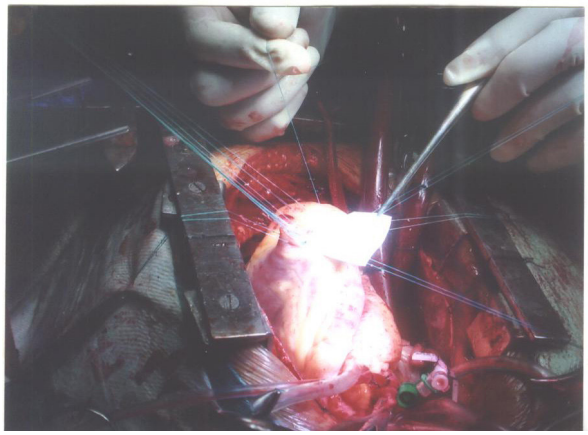


ANTERIOR VSR

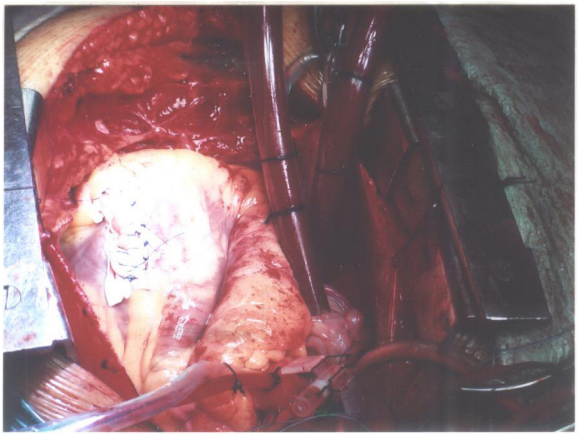
a.



b.

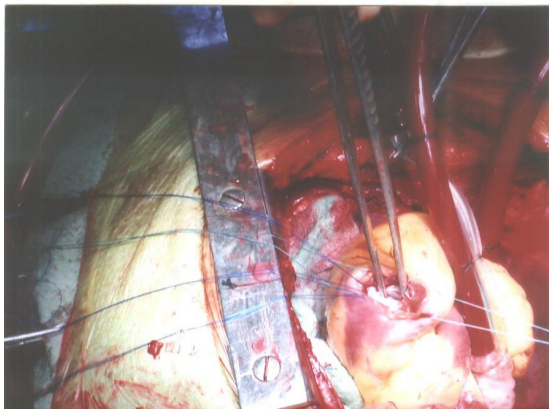


c.



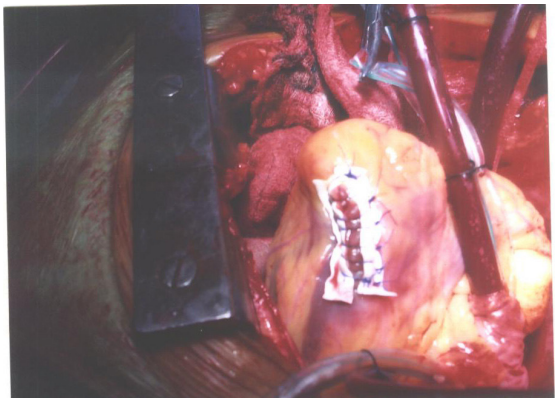
ANTERIOR VSR

a.



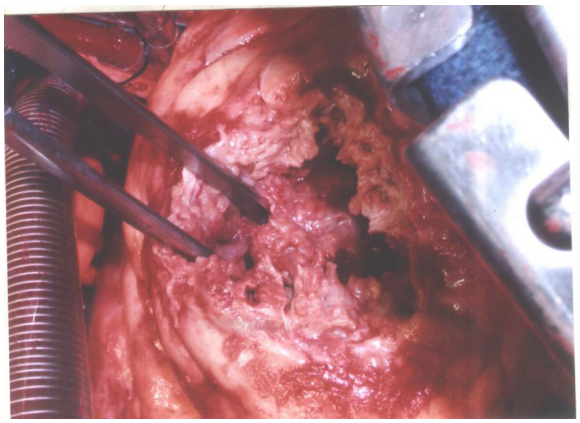
b.

c.

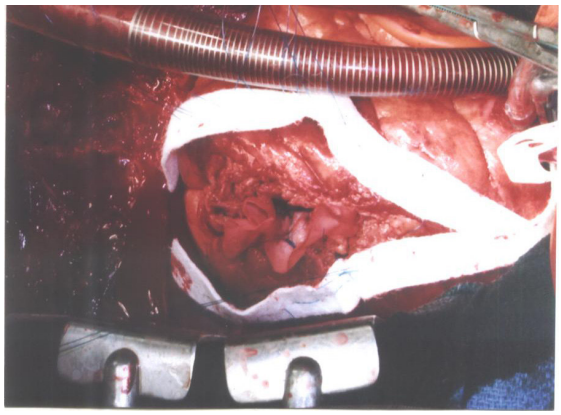


ACUTE VSR

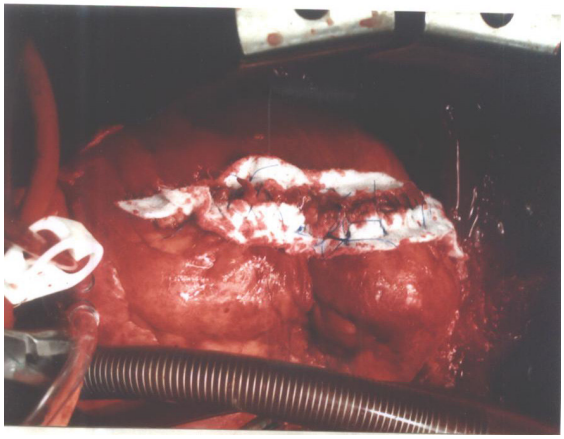
a.



b.

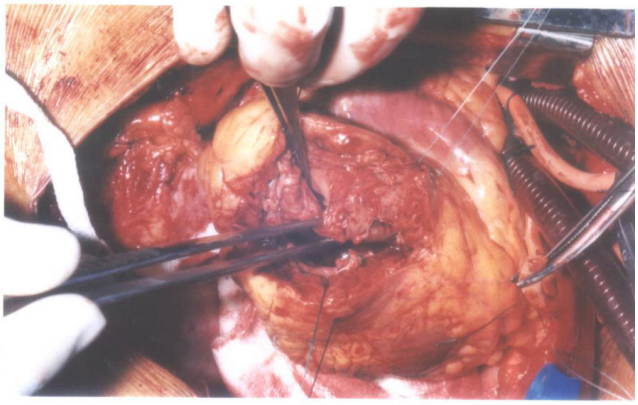


c.

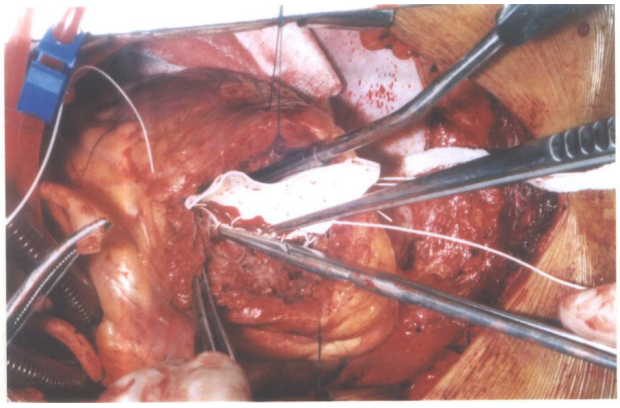


COMPLEX VSR

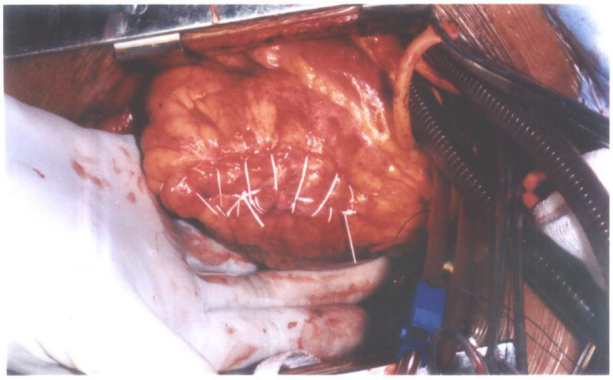
a.



b.

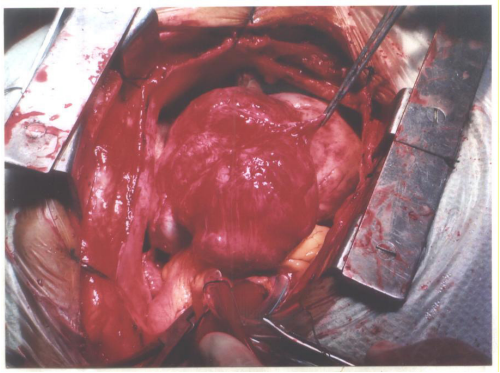


c.

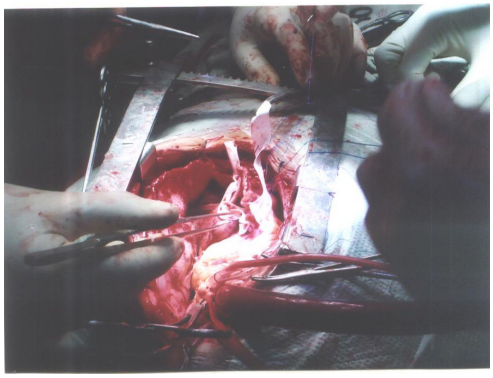


LV ANEURYSM

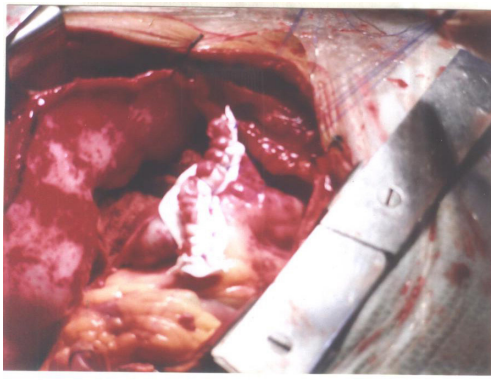
a.



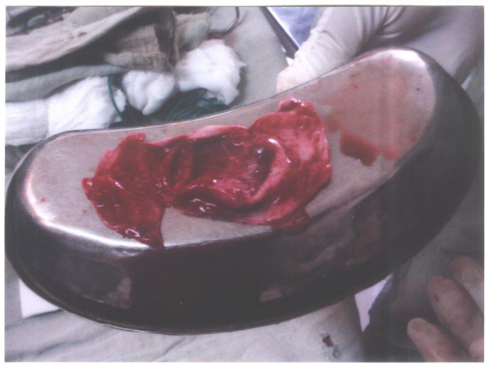
b.



c.

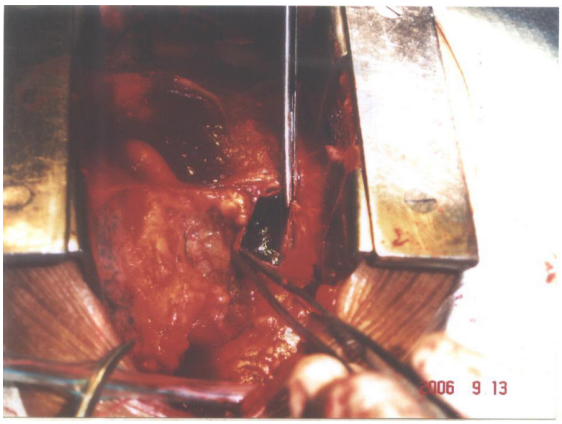


d.

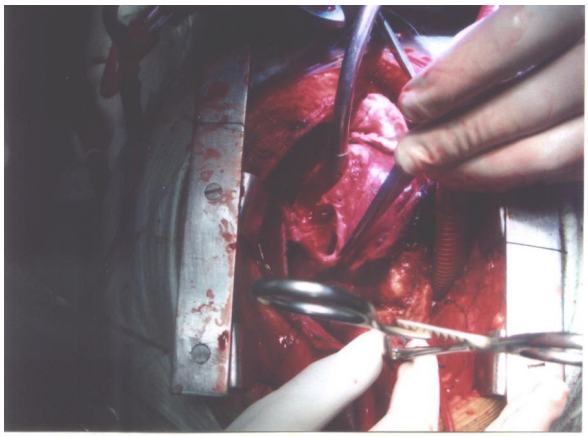


LV ANTERIOR WALL RUPTURE

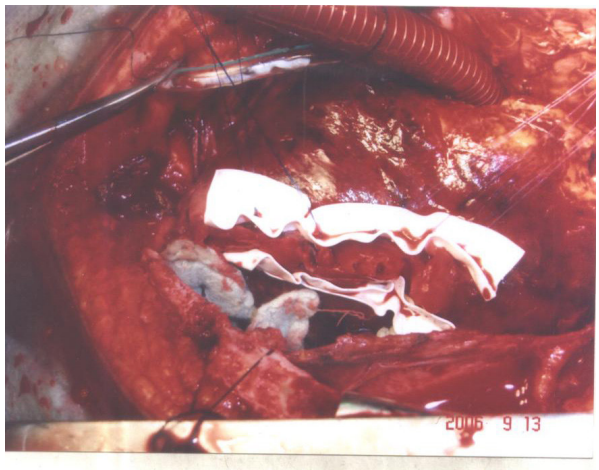
a.



b.



c.



OBSERVATION AND RESULTS

Year	*Total AMI*	*Total Death*	*Total VSR*	*VSR Death*	*VSR Operated*	*VSR Survived*	*VSR Post-op Death*
2002	475	35	3	2	1	1	–
2003	704	57	5	3	2	2	–
2004	964	83	9	5	4	3	1
2005	725	68	5	3	2	1	1
2006	1015	142	7	4	3	2	1
2007	364	29	4	2	2	2	–
	4247	**414**	**33**	**19**	**14**	**11**	**3**

VSR complicates 0.8% (33/4247) of AMI cases at our institution. Incidence remains same over the years.

VSR accounts for about 4.6% (19/414) of early death after AMI.

57.5% (19/33) of VSR patients managed medically died in ICU and 42.5% (14/33) survived to undergo surgical treatment.

AGE DISTRIBUTION

Age	*No*	%
30 - 40	-	-
41 - 50	3	21
51 - 60	10	82
61 - 70	-	-
71 - 80	1	7

SEX DISTRIBUTION

Sex	*No*	%
Male	9	64
Female	5	36

Age of patients with this complication ranges from 43-75 with mean age of 55 years. Maximum cases occurred in age group of 51-60 years.

It is twice more common in men with M:F ratio of 2:1 reflecting the increased incidence of CAHD in men.

CLINICAL PRESENTATION

Clinical Presentation	*No*	%
Stable	9	64
Cardiogenic shock	4	28
Multi organ failure	1	8

Most patients (9/14) were haemodynamically stable. 4 patients were in cardiogenic shock and 1 with multiorgan failure with raised BUN. All these 5 patients were stabilized with medical management and taken up for early surgery.

Case No	*Time between MI & diagnosis of VSR (days)*	*No. of attack*	*Risk Factors*	*ECG location of MI*	*VSD Location*
1	12	1	HT	Anterior	Anterior
2	9	1	–	Anterior	Anterior
3	5	1	–	Anterior	Anterior
4	15	1	S	Anterior	Anterior
5	8	2	DM/HT/S	Anterior	Anterior
6	3	1	HT/S	Anterior/ Inferior	Posterior
7	7	1	–	Anterior	Anterior
8	?	2	HT/S	Anterior	Anterior
9	?	1	HT	Anterior	Anterior
10	4	1	DM/HT/S	Inferior/ Lateral	Posterior
11	?	1	HT/S	Anterior	Anterior
12	2	1	–	Anterior	Anterior
13	8	1	HT	Anterior	Anterior
14	10	1	DM/HT	Inferior/ Lateral	Posterior

Time from infarction onset to diagnosis of VSR ranges from 2 days to 15 days.

Majority of patients 85% (12/14) developed VSR after their first attack.

15% (2/14) were diagnosed to have VSR during their 2nd attack evaluation. These patients had small VSR and remain clinically stable.

Risk Factor	***No***	**%**
Diabetics	3	21
Hypertension	9	64
Smoking	6	43
Previous MI	2	14

Hypertension and smoking are the common risk factors seen in patient developing post MI VSR.

Anterior wall myocardial infarction is seen in 78.5% (11/14) cases. Location of MI in ECG correlated well with the site of VSD intra operatively. In patients suspected to have post MI VSR clinically ECG can be used as a guide for localisation of VSR.

ANGIOGRAM

Case No	*LM*	*LAD*	*LCX*	*RCA*
1	Normal	100	–	–
2	Normal	100	–	–
3	Normal	100	–	–
4	Normal	Severe	70	70
5	Normal	100	70	60
6	Normal	60	–	100
7	Normal	100	–	–
8	Normal	100	60	–
9	Normal	100	–	–
10	Normal	80	60	100
11	Normal	100	–	–
12	–	–	–	–
13	Normal	100	–	60
14	–	–	–	–

Coronary angilogram was done for 12/14 patients. Angiogram was not done for two patients. One with free wall rupture and another patients with multiorgan failure.

10/12 patients had total occlusion of infarct related artery. The infarct artery was LAD in patients with anterior VSR and RCA in patients with posterior VSR.

Vessels involved	***No***	**%**
Single Vessel	7	58
Two Vessel	2	17
Three Vessel	3	25

58% of patients had Single vessel disease, 17% had Two vessel disease, and 25% had Three Vessel Disease. Poor colleteralisation is evident in all cases.

ASSOCIATED LESIONS:

Associated lesions:	*No*	%
Coronary Artery Stenosis	5	35
LV Aneurysm	3	21
Freewall Rupture	1	7

Multivessel coronary artery disease is seen in 5/12 patients.

Left ventricular aneurysm is seen in 3/14 patients who presented late after infarction.

One patient developed combined septal and left ventricular anterior wall rupture.

TIME INTERVAL BETWEEN INFARCTION AND SURGERY

Time interval	*No*	%
< 3 Weeks	4	28
3 to 6 Weeks	6	28
> 6 weeks	4	44

4/14 patients who presented with cardiogenic shock stabilized with medical management were operated within 3 weeks. Most of the stable patients are operated between 3-6 weeks. 3 patients with LV aneurysm presented late to our hospital and were treated more than 6 weeks time.

VSD MORPHOLOGY

Site	***No***	**%**
Anterior	11	79%
Posterior	3	21

Number	***No***	**%**
Single	13	93
Multiple	1	7

Type	***No***	**%**
Simple	12	86
Complex	2	14

Post Myocardial infarction septal rupture are most commonly located in anteroapical septum 79% as a result of full thickness anterior infarction. These anterior septal ruptures are caused by antero septal MI following occlusion of LAD. In 21% of cases rupture occurred in posterior septum following inferoseptal infarction which is usually due to dominant RCA.

VSR are single in 93% of cases and one patient (7%) had two separate VSD probably due to infarct extension.

86% of patients had simple through and through defect with well defined margins located anteriorly. Two patients (14%) had complex rupture i.e., irregular defect with indistinct margin located posteriorly.

APPROACH

Approach	*No*	%
Left Vetriculotomy	14	100
Right Vetriculotomy	-	-
Trans atrial	-	-

All VSR are approach through an incision over infarcted Myocardium in left vetricul. This trans infarct left vetriculotomy incision has various advantages. It avoids incision in normal myocardial area i.e., such collaterals are not disturbed and further ventricular distortion does not occur. Infarcted scarred area holds suture better, facilitates aneurysmorraphy if needed. Right vetriculotomy and transatrial approach are not used in our hospital.

Technique	*No*	%
Direct Closure	1	7
Patch Closure	13	93
Infarct Exclusion	-	-

Gortex patch is used to close VSD in 93 % of cases. In patient with small apical VSR direct placation technique was used.

Concomitant Procedures

Procedure	***No***	***%***
CABG	5	35
LV Aneurysm Repair	3	21
MVR	-	-
Infarctectomy	1	7

Simultaneous coronary revascularisation was done in 5 patients. Saphenous vein graft was used in all cases.

Aneurysmorraphy was done in 3 patients with LV aneurysm infarctectomy and patch closure was done in 1 patient with free wall rupture.

OUT COME

21 % 3/14 patients died post operatively.

2 Patients died due to suture line bleeding from friable myocardium and 1 patient died in immediate postoperative period due to low cardiac output.

Factors influencing surgical outcome

Preop NYHA Class	***Total No***	***Survived***	***Dead***	***Mortality***
I	-	-	-	-
II	3	3	-	-
III	6	5	1	16%
IV	5	3	2	40%

Risk Factors	***Total No***	***Survived***	***Dead***	***Mortality***
Diabetics	3	2	1	33%
Hypertension	9	6	3	33%
Smoking	6	3	3	50%
Previous MI	2	1	1	50%

Time from VSR to Surgery	***Total No***	***Survived***	***Dead***	***Mortality***
< 3 Weeks	4	2	2	50%
3 to 6 Weeks	6	6	-	0%
> 6 Weeks	4	3	1	25%

Location of MI and VSD	***Total No***	***Survived***	***Dead***	***Mortality***
Anterior	11	10	1	9%
Posterior	3	2	2	67%

Concomitant Procedure	***Total No***	***Survived***	***Dead***	***Mortality***
CABG	5	3	2	40%
Aneurysm Repair	3	2	1	33%

Preoperative condition of the patient is an important prognostic factor patient in NYHA Class III / IV represent severe form of disease with poor out come. History of previous MI and smoking are associated with increased mortality.

Early surgery with in 3 weeks has 50% mortality in our study. This is probably due to poor general condition of the patient who showed little improvement with medical management, non availability of IABP support at our institution, friable nature of infarcted myocardium failing to hold sutures with postop bleeding as occurred in two of our cases.

Posterior location of VSR is associated with 67 % mortality rate this is due to extensive infarction with poor myocardial function and frequent occurance of complex type of VSR.

Simultaneous CABG is associated with 40% mortality and aneurysm repair with 33% mortality.

CONCLUSION

VSR complicates 0.8 % of acute myocardial infarction.

It is more common in males between 51 to 60 years age, Hypertensive and Smoker, Experiencing First Heart Attack.

Most have anterior wall infarction with single vessel disease and completely occluded coronary artri and poor colletrals.

VSD is more common in anterior location, single, simple type and moderate size.

Preoperative condition, smoking history, posterior location of VSR, early surgery within 3 weeks, associated multi vessels CAD, are associated with increased postoperative mortality.

Delayed surgery after 3 weeks of medical stabilization increases success rate.

Concomitant CABG and aneurysm repair improved postoperative out come.

REFERENCES

1. Daggetr WM: Post infarction ventricular septal defect repair: retrospective thoughts and historical perspectives. Ann Thoracic Surg. 1990; 50:1006.

2. Latham PM: Lectures on Subjects Connected with Clinical Medicine Comprising Diseases of the Heart. London, Longman Rees, 1845.

3. Brunn F: Diagnostik der erworbenen ruptur derkammerscheidewand and des herzens. Wien Arch Inn Med 1923; 6:533.

4. Sager R: Coronary thrombosis: perforation of the infarcted inter ventricular septum. Arch Intern Med 1934; 53:140.

5. Cooley CIA, Belmonte BA, Zeis LB, Schnur 5: Surgical repair of ruptured interventricular septum following acute myocardial infarction. Surgery 1957; 41:930.

6. Euffler DB, Tapia FA, MeCormack LJ: Rupture of the ventricular myocardium and perforation of the interventricular septum complicating acute myocardial infarction. Circulation 1959; 20:128.

7. Payne WS, Hunt JC, Kirklin JW: Surgical repair of ventricular septal defect doe to myocardial infarction: report of a case. JAMA 1963; 183:603.

8. Sanders RJ, Kern WH, Blount SG: Perforation of the interventricular septum complicating myocardial infarction. Am Heart J 1956; 5 1:736.

9. Lee WY, Cardon L, Slodki SV: Perforation of infarcted interventricular septum. Arch Intern Med 1962; 109:135.

10. Dobell ARC, Scott HJ, Cronin RFP, Reid EAS: Surgical closure of interventricular septal perforation complicating myocardial infarction. J Thorac Cardiovasc Surg. 1962; 43:802.

11. Daicoff AR, Rhodes ML: Surgical repair of ventricular septal rupture and ventricular aneurysms. JAMA 1968; 203:457.

12. Allen P. Woodwark G: Surgical management of Post infarction ventricular septal defects. J Thorac Cardiovasc Surg 1966; 5 1:346.

13. Heimbecker RO, Lemire C, Chen C: Surgery for massive myocardial infarction. Circulation 1968; 11(suppl 2):3.

14. Iben AB, Pupello DF Stinson EB, Shumway NE: Surgical treatment of Post infarction ventricular septal defects. Ann Thorac Surg 1969;8:252.

15. Lojos TZ, Greene DG, Bunnell IL, et al: Surgery for acute myocardial infarction. Ann Thorac Surg 1969; 8:452.

16. Daggett WM, Burwell LR, Lawson DW, Austen WG: Resection of acute ventricular aneurysm and ruptured interventricular septum after myocardial infarction. N Engl J Med 1970; 283:1507.

17. Stinson EB, Becker J, Shumway NE: Successful repair of Post infarction ventricular septal defect and biventricular aneurysm. J Thorac Cardiovasc Surg 1969; 58:20.

18. Freeny PC, Schattenberg TT, Danielson GK, et al: Ventricular septal defect and ventricular aneurysm secondary to acute myocardial infarction. Circulation 1971; 43:360.

19. Daggett WM, Guyton RA, Mundth ED, et al: Surgery for postmyocardial infarct ventricular septal defects. Ann Surg 1977;186:260.

20. Daggett WM, Mundth ED, Gold HK, et al: Early repair of ventricular septal defects complicating inferior myocardial infarction. Circulation 1974; 50 (suppl 3):112.

21. Daggett WM: Surgical management of ventricular septal defects complicating myocardial infarction. World Surg 1978; 2:753.

22. Skillingon PD, Davies RH, Luff AJ, et al: Surgical treatment for infarct-related ventricular septal defects. J Thorac Cardiovasc Surg 1990; 99:798.

23. Lundberg S, Sodestrom J: Perforation of the interventricular septum in myocardial infarction: a study based on autopsy material. Acta Med Scand 1962; 172:413.

24. Hutchins GM: R9pture of the interventricular septum complicating myocardial infarction: pathological analysis of 10 patients with clinically diagnosed perforation. Am Heart J 1979; 97:165.

25. Kitamura S, Mendez A, KayJH: Ventricular septal defect following myocardial infarction: experience with surgical repair through a left ventriculotomy and review of the literature. J Thorac Cardiovase Surg.1971; 61:186.

26. Selzer A, Gerbode F, Keith Wi: Clinical, hemodynamic and surgical considerations of rupture of the ventricular septum after myocardial infarction. Am Heart J 1969; 78:598.

27. Mann JM, Robert WC: Acquired ventricular septal defect during acute myocardial infarction: analysis of 38 unoperated necropsy patients and comparison with 50 unoperated necropsy patients without rupture. Am] Cardiol 1988; 62:8.

28. Mallory GK, White PD, Salcedo-Salgar J: The speed of healing of myocardial infarction: a study of the pathologic anatomy in seventy cases. Am Heart] 1939; 18:647.

29. Silver MD, Butany J, Chiasson DA: The pathology of myocardial infarction and its mechanical complications, in David TE (ed): Mechanical Complications of Myocardial Infarction. Austin, TX, RG Landes, 1993; p 4.

30. Davies RH, Dawkins KD, Skillington PD, et al: Late functional results after surgical closure of acquired ventricular septal defect. J Thorac Cardiovasc Surg 1992; 106:592.

31. Daggett WM, Buckley MJ, Akins CW, et al: Improved results of surgical management of Post infarction ventricular septal rupture. Ann Surg 1982; 196:269.

32. Skehan JD, Carey C, Norrell MS, et al: Patterns of coronary artery disease in post-infarction ventricular septal rupture. Br Heart, 1989; 62:268.

33. Miller S, Dinsmore RE, Grenne RE, Daggett WM: Coronary, ventricular, and pulmonary abnormalities associated with rupture of the interventricular septum complicating myocardial infarction. Am] Radiol 1978; 131:571.

34. Hill JD, Lary D, Keith WJ, Gerbode F: Acquired ventricular septal defects: evolution of an operation, surgical technique and results. J Thorac Cardiovasc Surg 1975; 70:440,

35. Deville C, Fontan F, Chevalier JM, et al: Surgery of post-infarction ventricular defect: risk factors for hospital death and long-term results. EurJ Cardiothorac Surg 1991; 5:167.

36. Swithingbank jM: Perforation of the interventricular septum in myocardial infarction. Br Heart] 1959; 21:562.

37. Cummings RG, Reimer KA, Catliff R, et al: Quantitative analysis of right and left ventricular infarction in the presence of postinfarction ventricular septal defect. Circulation 1988; 77:33.

38. Weisman HF, Healy B: Myocardial infarct expansion, infarct extension, and reinfarction: pathophysiologic concepts. Prog Cardiovasc Dis 1987; 30:7 3.

39. David TE: Surgery for postinfarction ventricular septal defects, in David TE (ed): Mechanical Complications of Myocardial Infarction. Austin, TX, RG Landes, 1993; P 175.

40. Beranek JT: Hyaline degeneration of myocardium is implicated in the pathogenesis of postinfarction heart rupture. Cor Notes 1994; 9:3.

41. Edwards BS, Edwards WD, Edwards E: Ventricular septal rupture complicating acute myocardial infarction: identification of simple and complex types in 53 autopsied hearts. Am J Cardiol 1984: 54:1201.

42. Pfeffer MA, Braunwafd F: Ventricular remodeling after myocardial infarction: clinical observations and clinical implications. Circulation 1990; 81:1161.

43. Abrams D, Edilist A, Luria M, Miller A: Ventricular aneurysms. Circulation 1963; 27:164.

44. Fanapazir L, Bray CL, Dark jP: Right ventricular dysfunction and surgical outcome in poszinfarction ventricular septal defect. EurJ Cardiothorac Surg 1983, 4:155.

45. David TE, Dale L, Sun Z: Postinfarction ventricular septal rupture: repair by endocardial patch with infarct exclusion.] Thorac Cardiovasc Surg 1995; 110:1315.

46. Radford MJ, Johnson RA, Daggett WM, et al: Ventricular septal defect following myocardial infarction: factors affecting survival. Clin Res 1978; 26:262A.

47. Moore CA, Nygaard TW, Kaiser DL, et al: Postinfarction ventricular septal rupture: the importance of location of infarction and right ventricular Function in determining survival, Circulation 1986; 74:45.

48. Zehender M, Kasper V' Kauder E: Right ventricular infarction as an independent predictor of prognosis after acute inferior myocardial infarction. N EnglJ Med 1993; 328:98 1.

49. Anderson DR Adams S. Bhat A, Pepper JR: Postinfarction ventricular septal defect: the importance of site of infarction and cardiogenic shock ort outcome. Eur J Cardiotho rac Surg 1989; 3:554.

50. Campion BL, Harrison CE, Guiliani ER, et al: Ventricular septal defect after myocardial infarction. Ann Intern Med 1969; 70:25 1.

51. Vlodaver Z, Edwards JE: Rupture of ventricular septum or papillary muscle complicating myocardial infarction. Circulation 1977: 55:815.

52. Honey M, Belcher JR. Hasa M, Gibbons JRP: Case reports: successful early repair of acquired ventricular septal defect after myocardial infarction. Br Heart] 1967; 29:453.

53. Rawlins MO, Mendel 0, Braimbridge MV: Ventricular septal defect and mitral regurgitation secondary to myocardial infarction. BrHeartJ 1972; 34:323.

54. Taylor PH, Citron D5, Rohicsek F, Sanger PW: Simultaneous repair of ventricular septal defect and left ventricular aneurysm following myocardial infarction. Ann Thorac Surg 1965; 1:72.

55. Meister SG, Helfant RI-I: Rapid differentiation of ruptured inter-ventricular septum front acute nntral insufficiency. N EngI j Med 1972; 287:1024.

56. Hillis LD, Firth BG, Winnilord MD: Variability of right-sided cardiac oxygen saturations in adults with and without left-to-right intracardiac shunt. Am] Catthol 1986; 58:129.

57. Heiffila J, Karesoja M: Ruptured interventricular septum complicating acute myocarclial infarction. Chest 1974; 66:675.

58. Buckley MJ, Mundth ED, Daggett W[v1. et al: Surgical therapy for early complications of myocardial infarction. Surgery 1971; 70:8 14.

59. Harrison MR, MacPhail B, Gurfey JC, et al: Usefulness of color Doppler flow imaging to distinguish ventricular septal defect from acute mitral regurgitation complicating acute myocardial infarction. Am J Cardiol 19b9: 64:697.

60. Smyllie JH, Sutherland GR, Geuskens R, et at: Doppler color flow mapping in the diagnosis of ventricular septal rupture and acute mitral regurgitation after myocardial infarction. JAm Coil Cardiol 1990; 15:1455.

61. Fortin DF, Sheikh KH, Kisslo 1: The utility of echocardiography in the diagnostic strategy of postinfarction ventricular septal rupture: a comparison of two-dimensional versus Doppler color flow imaging. Am Heart J 1991; 121:25,

62. Blanche C, Khan SS, Matloff JM, et al: Results of early repair of ventricular septal defect after an acute myocardial infarction. J Thorac Cardiovasc Surg 1992; 104:961,

63. Matsui K, Kay JH, Mcndez M, et al: Ventricular septal rupture secondary to myocardial infarction: clinical approach and surgical results. JAMA 1981; 245:1537.

64. Kaplan MA, Harris CN, Kay JH, et al: Postinfarctional septal rupture: clinical approach and surgical results. Chest 1976; 69:734.

65. Oyamada A, Queen FB: Spontaneous rupture of the interventricular septum following acute myocardial infarction with some clinico-pathologic observations on survival in five cases [unpublished]. 1961.

66. Berger TJ, Blackstone El-I, Kirklin JW: Postinfarction ventricular septal defect, in Kirk tin JW Barratt-Boyes BG (eds): Cardiac Surgery. New York, Churchill Livingstone, 1993; p 403.

67. Lemery R, Smith HC, Giuliani ER, Gcrsh BJ: Prognosis in rupture of the ventricular septum after acute myocardial infarction and role of early surgical intervention. Am J Cardiol 1992; 70:147.

68. Menon V, Webb J, Hillis D, et at: Outcome arid profile of ventricular septal rupture with cardiogentic shock after myocardial infarction: a report from the SHOCK Trial Registry J Am Coil Cardiol 2000; 36:1010.

69. Kay HR: In discussion of Daggeti WM: Surgical management of ventricular septal defects complicating myocardial infarction. World J Surg 1978; 2:753.

70. Heitmiller R, Jacobs ML, Daggett WM: Surgical management of postinfarction ventricular septal rupture. Ann Thorac Surg 1986; 41:683.

71. Estrada-Quintero T, Uretsky BE Murali S, Hardesty RL: Prolonged intraaortic balloon support for septal rupture after myocardial infarction. Ann Thorac Surg 1992; 53:335.

72. Baillot R, Pelletier C, Trivino-Marin j, Castonguay Y: Postinfarction ventricular septal defect: delayed closure with prolonged mechanical circulatory support. Ann Tliorac Surg 1983; 35:138.

73. Muehrcke DD, Daggett V'/M, Buckley MJ, et al: Postinfarct ventricular septal defect repair: effect of coronary artery bypass grafting. Ann Thorac Surg 1992; 54:876.

74. 74. Gaudiani VA, Miller DC, Oyei PE, et al: Post-infarction ventricular septal defect: an argument for early operation. Surgery 1981, 89:48.

75. Gold HK, Leinbach RC, Sanders CA, et al: lntra-aortic balloon pumping for ventricular sepial defect or mitral regurgitation complicating acute myocardial infarction. Circulation 1973; 47:1191.

76. Montoya A: Ventricular septal rupture secondary to acute myocardial infarction, in Pifarre R (ed): Cardiac Surgery: Acute Myocardial Infarction and Its Complications. Philadelphia, Hanley & Belfus, 1992; p 159.

77. Scanlon PJ, Monatoya A, Johnson SA: Urgent surgery for ventricular septal rupture complicating myocardial infarction. Circulation 1985; 72 (suppl 2):185.

78. Loisance DY, Cachera JP, Poulain H, et al: Ventricular septal defect after acute myocardial infarcion. J Thorac Cardiovasc Surg 1980; 80:61.

79. Babb JD, Waldhauscn JA, Zeus R: Balloon induced right ventricular outflow obstruction: a new approach to control of acute interventricular shunting alter myocardial infarction in canines and swine. Circ Res 1977; 40:372.

80. Meyns B, Vanermen H, Vanhaecke J, et al: Hemopump fails as bridge to transplantation in postinfarction ventricular septal defect. J Heart Lung Transplant 1994; 13:1133.

81. David H, HunterJA, Najafi H, et al: Left ventricular approach for the repair of ventricular septal performation and infarctectomy. J Thorac Caniovasc Surg 1972; 63:14.

82. Daggett WM, Randolph JD, Jacobs ML, et al: The superiority of cold oxygenated dilute blood cardioplegia. Ann Thorac Surg 1987;43:397.

83. Hendren WG, O'Keefe DD, Geffin GA, et al: Maximal oxygenation of dilute blood cardioplegia solution. Ann Thorac Surg 1994; 58:1558.

84. David TE: Surgical treatment of postinfarction ventricular septal rupture. AustralasJ Card Thorac Surg 1991; 1:7.

85. Weisel RD: Myocardial protection during surgery for mechanical complications of myocardial infarction, in David TE (ed): Mechanical Complications of Myocardial Infarction. Austin, TX, RG Landes, 1993; p 120.

86. Torchiana DF Geffln GA, O'Keefe DD, Daggett WM: Cardioplegia for repair of postinfarction ventricular septal defect, in Engleman RM, Levitsky S, (eds): A Textbook of Cardioplegia for Difficult Clinical Problems. Mount Kisco, NY, Futura, 1992; p 115.

87. Rosenkranz ER, Buckberg GD, Mulder DG, Laks H: Warm induction of cardioplegia with glutamate-enriched blood in coronary patients with cardiogenic shock who are dependent on inorropic drugs and intraaortic ballon support: initial experience and operative strategy. J Thorac Cardiovasc Surg 983; 86:507.

88. Teoh KH, Christakis GT Weisel RD, et al: Accelerated myocardial metabolic recovery with terminal warm blood cardioplegia. J Thorac Cardfovasc Surg 1986; 91:888.

89. Shumacker I-I: Suggestions concerning operative management of postinfarction ventricular septal defects. J Thorac Cardiovasc Surg 1972; 64:452.

90. Dor V, Saab M, Coste P, et al: Left ventricular aneurysm: a new surgical approach. Thorac Cardiovasc Surg 1989; 37:11.

91. Cooley DA: Repair of the difficult ventriculotomy. Ann Thorac Surg 1990; 49:150.

92. Cooley DA: Repair of post infarction ventricular septal defect. J Canl Surg 1994; 9:427.

93. Alvarez JM, Brady PW, Ross DE: Technical improvements in the repair of acute postinfarction ventricular septal rupture. J Card Surg 1992; 7:198.

94. daSilva JP, Cascudo MM, Baumgratz JF, et al: Post infarction ventricular septal defect: an efficacious technique for early surgical repair. J Thorac Cardiovasc Surg 1989; 97:86.

95. Tashiro T, Todo K, Haruta Y, et al: Extended endocardial repair of postinfarction ventricular septal rupture: new operative technique modification of the komeda-david operation. j Card Surg 1994;9:97.

96. Usui A, Murase M, Maeda M, et al: Sandwich repair with two sheets of equine pericardial patch for acute posterior 1ost- infarction ventricular septal defect. Eur J Cardiothrac Surg 1993; 7:47.

97. Fujimoto K, Kawahito K, Yamaguchi A, et al: Percutaneous extra-corporeal life support for treatment of fatal mechanical complications associated with acute myocardial infarction. Art if Organs 2001; 25: 1000.

98. Landzberg MJ, LockJE: Transcatheter management of ventricular septal rupture after myocardial infarction. Semin Thorac Cardiovasc Surg 1998; 10:128.

99. Lock JE, Block PC, McKay RG, ét al: Transcathemxr closure of ventricular septal defects. Circulation 1988; 78:361.

100. Hachida M, Nakano H, Hirai M, Shi CY: Percutaneous transaortic closure of postinfarction ventricular septal rupture. Ann Thorac Surg 1991; 51:655.

101. Abhyankar A, Jagtap P: Post-infarction ventricular septal defect: percutaneous transvenous closure using a Swan-Ganz catheter. Cathet Cadiovasc Interi 1999; 47:208.

102. Aliahadi D, Roland C, Pett 5, et al: Percutaneous cardiopulmonary support for the management of catastrophic mechanical complications of acute myocardial infarction. Cathet Cardiovasc Diagn 1996; 37:223.

103. Ksheury V, Salerno C, Bank A: Risk of left ventricular assist device as a bridge to heart transplant following postinfarction ventricular septal rupture.J Card Surg 1997; 12:93.

104. Meyns B, Vanermen H, Vanhaecke J, et al: Hemopump fails as bridge to transplantation in postinfarction ventricular septal defect.] Heart Lung Transplant 1994:13:1133.

105. Loisance DP, LordezJM, Deleuze PH, et al: Acute postinfarction septal rupture: long-term results. Ann Thorac Surg 1991; 52:474.

106. Piwnica A, Menasche P, Beaufils P, Julliard JM: Long-term results of emergency surgery for postinfarction ventricular septal defect. Ann Thorac Surg 1987; 44:274.

107. Weintraub RM, Thurer RL, Wei J: Repair of postinfarction ventricular septal defect in the elderly: early and long term results. J Thorac Cardiovasc Surg 1983; 85:191.

108. Marchetti A, Wechsler AS. Pharmacologic intervention for acute low cardiac output [abstract]. Adv Card Surg 1996; 7:2 1.

109. D'Ambra MN, LaRaia PJ, Philbin DM, et al: Prostaglandin Ej: a new therapy for refractory right heart failure and pulmonary hypertension after mitral valve replacement.J Thorac Cardiovasc Surg 1985; 89:567.

110. Rich GF, Murphy GDJr, Roos CM,Johns RA: Inhaled nitric oxide: selective pulmonary vasodilatation in cardiac surgical patients. Anesthesiology 1993; 78: 1028.

111. Pae WE Jr, Pierce WS, Sapirstein JS: Intra-aortic balloon counterpulsation, ventricular assist pumping, and the artificial heart, in Bauc AE, Geha AS, Hammond GL, Laks H, Naunheim KS, (eds): GIenni Thoracic and Cardiovascular Surgery. Stamford, CT, Appleton St Lange, 1996; p 1825.

112. Sundt TM 3rd, Kouchoukos NT, SaffitzJE, et al: Renal dysfunction and intravascular coagulation with aprorinin and hypothermic circulatory arrest. Ann Thorac Surg 1993; 55:1418.

113. Seguin JR, Frapier JM, Colson P, Chaptal PA: Fibrin sealant for early repair of acquired ventricularseptal defect. j Thorac Cardiovase Sarg 1992; 104:748.

114. Fabiani J-N, Jebara VA, Deloche A, et al: Use of surgical glue without replacement in the treatment of type A aortic dissection. Circulation 1989; 80:264.

115. Baldwin RT, Cooley DA: Mechanical support for intraventricular decompression in repair of left ventricular disruption. Ann Thorac Surg 1992; 54:176.

116. Saksena 5, Rothbart ST, Shah Y: Clinical efficacy and electrophar macology of continuous intravenous amiodarone infusion and chronic oral amiodarone in refractory ventricular tach)'cardi Am - J Cardiol 1984; 54:347.

117. Komeda M, Fremes SE, David TE: Surgical repair of the postinfarcLion ventricular septal defect. Circulation 1990; 82(suppl 4):243.

118. Radford MJ,Johnson RA, Daggett WM, et al: Ventricular septal rupture: a review of clinical and physiological features and an analysis of survival. Circulation 1981; 64:545.

119. Cox FF, Morshuis WJ, Plokker HWT, et al: Early mortality after surgical repair of post-infarction ventricular septal rupture: importance of rupture location. Ann Thorac Surg 1996; 61:] 752.

120. Jones MT Schofield PM, DarkJF: Surgical repair of acquired ventricular septal defect: determinants of early and late outcome. J Thorac Cardiovasc Surg 1987; 93:680.

121. Dalrymple-Hay MJR, Monro JL, Livesey SA, Lamb RK: Postinfarction ventricular septal rupture: the Wessex experience. Sernin Thorac Cardiovasc Surg 1998; 10:111.

122. Hill JD, Stiles QR: Acute ischernic ventricular septal defect Circulation 1989; 79(suppl 1):1 12.

123. Ross D: Overlay patch technique for ventricular septal defect repair. AustralasJ Card Thorac Suig 1992; 1:11.

124. Cooley DA: Postinfarction ventricular septal rupture. Sentin Thorae Cardiovasc Surg 1998; 10:100.

125. Rousouj A, Engelman RM, llreyer RH, et al: Transatrial repair of postinfarction posterior ventricular septal defect. Ann Thorac Surg. 1987; 43:665.

126. Filgueira JL, Battistcssa SA, Estable H, et al: Delayed repair of an acquired posterior septal defect through a right atrial approach.

Case No.	***1***	***2***	***3***	***4***	***5***	***6***	***7***	***8***	***9***	***10***	***11***	***12***	***13***	***14***
Age (Years)	57	52	56	46	53	57	60	54	53	48	55	75	43	60
Sex	F	M	F	M	M	M	F	M	F	M	M	F	M	M
IP No.	560217	600878	615093	641144	663355	690642	691167	683684	712529	804841	792429	836575	015003	029616
Year	2002	2003	2003	2004	2004	2004	2004	2005	2005	2006	2006	2006	2007	2007
Clinical Presentation (NYHA Class)	III	II	IV	II	III	IV	II	IV	III	IV	III	IV	III	IV
Time Between MI & Diagnosis (days)	12	9	5	15	8	3	7	?	?	4	?	2	8	10
No of attack	1	1	1	1	2	1	1	2	1	1	1	1	1	1
Risk Factors	HT	-	-	S	DM/HT/S	HT/S	-	HT/S	HT	DM/HT/S	HT/S	-	HT	DM/HT
ECG site of MI	AWMI	AWMI	AWMI	AWMI	AWMI	IWMI/ AWMI	AWMI	AWMI	AWMI	IWMI/ LWMI	AWMI	AWMI	AWMI	IWMI/ LWMI
Angio No. of Vessles involved	1VD	1VD	1VD	3VD	3VD	2VD	1VD	2VD	1VD	3VD	1VD	-	2VD	-
Associated lesions	-	-	-	-	-	-	-	LVA	LVA	-	LVA	FWR	-	-

Case No.	***1***	***2***	***3***	***4***	***5***	***6***	***7***	***8***	***9***	***10***	***11***	***12***	***13***	***14***
Time between MI & Surgery (Weeks)	3-6	3-6	<3	3-6	>6	<3	3-6	>6	>6	<3	>6	<3	3-6	3-6
VSD Location	Anterior	Anterior	Anterior	Anterior	Anterior	Posterior	Anterior	Anterior	Anterior	Posterior	Anterior	Anterior	Anterior	Posterior
Approach	LV	LV	LV	LV	LV	LV	LV	LV	LV	LV	LV	LV	LV	LV
Technique	PC	DC	PC	PC	PC	PC	PC	PC	PC	PC	PC	PC	PC	PC
Concomitant Procedure	-	-	-	CABG	CABG	-	-	CABG/ LVAR	-	CABG	-	-	CABG	-
Hospital Survival	Yes	Yes	Yes	Yes	Yes	No	Yes	No	Yes	No	Yes	Yes	Yes	Yes
Post of NYHA Class	II	I	II	II	II	-	I	I	II		II	III	II	II

HT - Hypertension, DM - Diabetes Mellitus, S - Smoking, LVA - Left Ventricle Aneurysm, LVAR - Left Ventricle Aneurysm Repair, FWR - Free Wall Rupture, VD - Vessel Disease.